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ROYAL COMMISSION OF INQUIRY INTO CERTAIN
DEATHS AT THE HOSPITAL FOR SICK CHILDREN AND
RELATED MATTERS.

Hearing held
8th floor
180 Dundas Street West
Toronto, Ontario

Row: X
(Scott)

The Honourable Mr. Justice S.G.M. Grange

Commissioner

P.S.A. Lamek, Q.C.

Counsel

E.A. Cronk

Associate Counsel

Thomas Millar

Administrator

Transcript of evidence
for

August 16, 1983

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ROYAL COMMISSION OF INQUIRY INTO CERTAIN
DEATHS AT THE HOSPITAL FOR SICK CHILDREN
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Hearing held on the 8th Floor,
180 Dundas Street West, Toronto,
Ontario, on Tuesday, the 16th
day of August, 1983.

- - - - -

THE HONOURABLE MR. JUSTICE S.G.M. GRANGE - Commissioner
THOMAS MILLAR - Administrator
MURRAY R. ELLIOT - Registrar

- - - - -

APPEARANCES:

P.S.A. LAMEK, Q.C.)	Commission Counsel
E. CRONK)	
M. HAYES)	
T.C. MARSHALL, Q.C.)	Counsel for the Attorney-
D. HUNT)	General and Solicitor
L. CECCHETTO)	General of Ontario (Crown
	Attorneys and Coroner's Office)
I.G. SCOTT, Q.C.)	Counsel for The Hospital
R. BATTY)	for Sick Children
M. THOMSON)	
B. PERCIVAL, Q.C.)	Counsel for The Metropolitan
D. YOUNG)	Toronto Police
W.N. ORTVED)	Counsel for numerous Doctors
K. CHOWN)	at The Hospital for Sick
	Children
E. McINTYRE	Counsel for the Registered
	Nurses' Association of Ontario
	and 35 Registered Nurses at
	The Hospital for Sick Children



APPEARANCES: (Continued)

H. SOLOMON	Counsel for the Ontario Association for Registered Nursing Assistants
J. SOPINKA, Q.C.	Counsel for Susan Nelles - Nurse
G.R. STRATHY) E. FORSTER) P. RAE)	Counsel for Phyllis Trayner - Nurse
B. JACKMAN	Counsel for Mrs. M. Christie - R.N.A.
J.A. OLAH	Counsel for Janet Brownless - R.N.A.
M. MANNING, Q.C.	Counsel for Mr. & Mrs. Gosselin, Mr. & Mrs. Gionas, Mr. & Mrs. Inwood, Mr. & Mrs. Turner, Mr. & Mrs. Lutes and Mr. & Mrs. Murphy (parents of deceased children)
G.R. SOLOMON	Counsel for Mr. & Mrs. Hines, parents of deceased child Jordan Hines)
F.J. SHANAHAN	Counsel for Mr. & Mrs. Dominic Lombardo (parents of deceased child Stephanie Lombardo); and Heather Dawson (mother of deceased child Amber Dawson)
J. SHINEHOFT	Acting for Lorie Pacsai and Kevin Garnet (parents of deceased child Kevin Pacsai)



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/EMT/ak

1
2 ---Upon commencing at 10:00 a.m.

3 MR. SOPINKA: Mr. Commissioner, I
4 don't know whether this seating plan is designed to
5 promote settlement, but I object to sitting next to
6 the Attorney-General.

7 MR. PERCIVAL: He doesn't have to
8 because my friends have arrived so he has to go
9 some place else.

10 MR. SCOTT: The problem is,
11 Mr. Sopinka, everybody objects to sitting next to
12 the Attorney-General but it had to be somebody.

13 THE COMMISSIONER: Well, I think we
14 should all be greatly honoured by his attendance today.

15 Obviously we are just trying to work
16 our way out, and all complaints will be heard in the
17 course of the day or at the end of the day or any
18 other time as to seating. I see now that Mr. Hunt
19 is here with no seat provided for him.

20 If you think this is unfortunate all
21 you have to do is contemplate what would have
22 happened if we had been up on the 21st floor where
23 they wanted to put us.

24 Well now, Mr. Lamek, you have a few
25 more questions, I believe, have you not?

MR. LAMEK: Mr. Commissioner, before
we begin again with Dr. Rowe I think we should perhaps



1
2 first for the record thank the Chairman of the
3 Municipal Board for permitting us the use of the
4 room and at the same time, sir, acknowledge your
5 advocacy skills in persuading him to do that.

6 Second, in the course of the two
7 weeks since we were last sitting, Mr. Shinehoft
8 has kindly provided to us his copy of the Pacsai
9 material, and replacement pages of some portions of
10 that chart have been prepared and distributed; in
particular, pages 33, 42, 45, 46 and 77.

11 As I say, replacement pages have been
12 distributed to counsel, sir, and perhaps at the end
13 of the day we can retrieve the exhibit copy and
14 replace them in that as well.

15 THE COMMISSIONER: What is the number
16 of that exhibit?

17 MR. LAMEK: The Pacsai exhibit?
18 I am sorry.

19 THE REGISTRAR: The Hospital record?

20 MR. LAMEK: Yes.

21 THE REGISTRAR: 106.

22 MR. LAMEK: 106. Thank you.

23 Next, Mr. Commissioner, the Hospital
24 has furnished to us copies of final autopsy reports
25 that do not appear in the bound Hospital charts that



1
2 have already been marked as exhibits.

3 We have had those prepared in a
4 separate binder with numbered pages, and I would ask
5 that that binder of final autopsy reports be the next
6 exhibit, and copies of that binder have been provided
7 to counsel.

8 THE COMMISSIONER: Exhibit 124, and
9 will you tell me which babies?

10 MR. LAMEK: Yes, sir. They are
11 Woodcock, Perreault, Taylor, Dawson, Hoos, Turner,
12 Shrum, Monteith, Velasquez, Gage, McKeil, Volk,
13 Lutes, Onofre, Gosselin, Belanger and Floryn.

14 THE COMMISSIONER: Thank you. That
15 is the final autopsy report for all the children ---

16 MR. LAMEK: Who were autopsied, yes.

17 THE COMMISSIONER: Yes. All right.

18 ---EXHIBIT NO. 124: Final Autopsy Report re Babies
19 Woodcock, Perreault, Taylor,
20 Dawson, Hoos, Turner, Shrum,
21 Monteith, Velasquez, Gage,
22 McKeil, Volk, Lutes, Onofre,
23 Gosselin, Belanger and Floryn.

24 MR. LAMEK: Mr. Commissioner, at
25 the end of our last hearing I had thought I had
completed my examination of Dr. Rowe, and I have
spoken to Mr. Scott, and with your permission, sir,
I have just a very few more questions of Dr. Rowe



1
2 with respect to a matter of which I learned only
3 last week.

4 THE COMMISSIONER: Yes.

5 MR. LAMEK: It will not take very
6 long, and perhaps we could ask Dr. Rowe to come back.

7 THE COMMISSIONER: Yes. All right.

8 MR. SCOTT: You couldn't save these
9 for reply?

10 MR. LAMEK: No. I think everybody
11 should know about it.

12 MR. ORTVED: If I may just have your
13 indulgence, Mr. Commissioner?

14 THE COMMISSIONER: Yes.

15 MR. ORTVED: Thank you, Mr. Commissioner.

16 DR. RICHARD DESMOND ROWE, Resumed

17 DIRECT EXAMINATION BY MR. LAMEK: (Continued)

18 Q. Dr. Rowe, first to complete
19 the matters that we were talking about when last we
20 met, you had given to me at the close of your
21 evidence a list of six names in addition to that of
22 Justin Cook, and those were the children who, as I
23 understood you, and subject to the resolution of any
24 pharmacological debate and dispute that may exist,
25 were those who in your judgment were most likely to
have died as a result of a digoxin overdose.



1
2 I understand that there is an additional
3 name that you omitted to give me in that list.

4 A. Yes. There is, Mr. Lamek.
5 That is the name of Lombardo.

6 Q. Yes.

7 A. And I appreciate the
8 opportunity to bring that in.

9 Q. Not at all. Thank you very
10 much, Doctor.

11 Now, Doctor, one thing has come to my
12 attention since last we met and I think we can deal
13 with it very rapidly. We discussed in the course
14 of your evidence in chief Mortality and Morbidity
15 Conferences that were held in September, 1980, and then
16 the meeting that was held in January, 1981, ~~and~~
17 following a review of some twenty deaths.

18 A. Yes.

19 Q. And at that stage as I under-
20 stood all ward deaths prior to December 31, 1980 had
21 been reviewed by your group?

22 A. Yes.

23 Q. Now I understand, Doctor, that
24 you had scheduled a further review to be done with
25 respect to deaths from and after January 1st, 1981,
and that that review was scheduled to begin on Monday,



1
2 March 21st, 1981.

3 A. No, I don't believe.

4 Q. You don't have a recollection?

5 A. No.

6 Q. You have told us that the
7 police investigation which, of course, was set in
8 train on the weekend of March 21 and 22 ---

9 A. Yes.

10 Q. --- overtook any investigations
11 by the Hospital?

12 A. Yes.

13 Q. But you have no recollection
14 of having arranged for a further review of the 1981
15 deaths to take place at the end of March?

16 A. No, not prior to the end of
17 the time of that weekend. I think at the end of
18 the weekend obviously there was going to have to be
19 examination of issues in connection with those deaths,
20 but that was taken by the police study.

21 Q. Do you have any recollection
22 on March 21, which I believe was the day when you
23 met with the coroners?

24 A. Saturday.

25 Q. Saturday the 21st would be
the day.



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A. Saturday the 21st would be
the day.

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Q. Do you have any recollection
at that time of saying to Dr. Teperman that in fact
a review had been arranged of the post January 1
deaths, and was to begin on the following Monday?

8

9

A. I can't recall.

10

11

Q. Then there is not much point
in my asking you any questions about that, Doctor.

12

13

A. No. I'm sorry.

14

15

MR. LAMEK: Thank you, Doctor.

16

THE COMMISSIONER: Yes. Thank you.

17

Mr. Scott?

18

19

MR. SCOTT: I think I will come
from here, Mr. Commissioner, it will be easier,
because I am going to need some help.

20

21

THE COMMISSIONER: Yes. All right.
Whatever is convenient.

22

23

MR. SCOTT: First of all,

24

Mr. Commissioner, I have placed on the ---

25

THE COMMISSIONER: I am not sure -
I can hear you. Can everyone hear you?

MR. SCOTT: This is not usually a
problem for me.

THE COMMISSIONER: No. But I am



1
2 just wondering whether that microphone is actually
3 working.

4 MR. SOPINKA: Well, I can't hear,
5 but I am not listening.

6 MR. SCOTT: Not yet.

7 THE COMMISSIONER: You don't know.
8 Well, I think it is probably working.
9 Let's try and if anyone can't hear just raise his
10 hand and we will do something about it.

11 The acoustics ^{are} ~~is~~ supposed to be very
12 good in here. I don't know whether they are or not.

13 MR. SCOTT: They are excellent,
14 almost everybody is inaudible, including the witness
15 and the Commissioner.

16 I should say, Mr. Commissioner, that
17 I put on the board what is really a series of graphs
18 which I undertake to prove when my opportunity, some
19 time late next year, comes to call evidence, but
20 because I may want to ask some questions about it I
21 tender it now as an exhibit, and perhaps it should
22 be given a number to be proved. And I want to explain
23 to you what it is and as I say, I undertake to prove
24 in due course the manner in which it has been
25 accomplished.

The data for the graph is based on the



1
2 monthly death reports which are compiled by the
3 census control clerk at the Hospital. Those monthly
4 reports are based upon a daily census sheet which
5 is completed for each ward or place in the Hospital
6 by the head nurse in charge of that ward or place,
7 and if a patient has died the census control clerk
8 checks for the last ward where the patient was alive
9 in order to get the geographical place of death.
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2 Now, in the left-hand margin, you
3 will see --

4 THE COMMISSIONER: Before we get
5 any further, you want to give it a number, I under-
6 stand?

7 MR. SCOTT: Yes.

8 THE COMMISSIONER: Exhibit 125.

9 --- EXHIBIT NO. 125: Mortality Chart.

10
11 THE COMMISSIONER: All right, the
12 left-hand side.

13 MR. SCOTT: Q. The left-hand side
14 are the number of deaths rated in multiples of five
15 and numbered in multiples of ten from zero to fifty.
16 Along the bottom, indicated by each point on the
17 measured line, are months from January 1, 1976 to
18 December 31, 1982. 'J' indicates January, the start-
19 ing point of each year. The black line at the top
20 of the page is the total deaths per month for that
21 period that occurred anywhere in the hospital.

22 So, at the top line, with the peaks
23 and valleys that it reveals, is a monthly accounting
24 of how the total number of deaths in the hospital
25 varied, and you will see, in examining that, that there



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are dots which represent the months and there are peaks and valleys and so on illustrated on that chart.

I am told that there is an orange line, and I don't see the orange line - it's a brown line. What is the brown line, neonates?

Yes, the brown line is, as you see, Ward 7G, which is the neonate ward.

THE COMMISSIONER: I'm sorry, which is the brown line?

MR. SCOTT: Here (indicating).

THE COMMISSIONER: No. I know what it is but what does it represent?

MR. SCOTT: You know what it looks like, you know what brown looks like.

THE COMMISSIONER: Yes. But what does it represent?

MR. SCOTT: It represents the neonate ward, which is --

THE COMMISSIONER: You have told us the brown line, but it seems to be over a -- oh, there is a yellow line as well?

MR. SCOTT: Well, we're coming to that.

THE COMMISSIONER: Yes. All right.

MR. SCOTT: The brown line is the



1
B3 2 neonate ward, No. 7G, and you will recall - I think
3 it is in evidence - that that is a neonatal intensive
4 care unit in which the age limit essentially is less
5 than one month of age.

6 Again, the number of deaths in that
7 ward is plotted on a monthly basis, and you will see
8 the degradations from month to month over this
9 period.

10 The next line is the all-cardiac
11 line.

12 THE COMMISSIONER: And that colour
13 is?

14 MR. SCOTT: And that colour is red -
15 it used to be orange - and that records, on a monthly
16 basis, all cardiac deaths - I think I've got the
17 cardiac line - yes, all cardiac deaths which occurred
18 in the hospital, and that will include cardiac deaths
19 in 4A and 4B. Again, it is plotted out on a monthly
20 basis.

21 The purple line --

22 THE COMMISSIONER: I take it the
23 only places, though, they could be would be, presumably,
24 in the operating room or in the intensive care unit?

25 MR. SCOTT: No. I will come to that.

MR. MANNING: I'm sorry, Mr.



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Commissioner, I can't hear you back here.

THE COMMISSIONER: Well, they promised me that this - no matter what I did to it - would record what's going on, but it obviously doesn't. We will have to work on that.

Can you hear me now?

MR. MANNING: Yes.

THE COMMISSIONER: I guess I just start to mutter. My trouble is, when I'm not too sure what I'm saying, I mutter and, when I have some idea what it's all about, I yell!

All right now.

MR. SCOTT: I just want to make a note of that!

THE COMMISSIONER: The cardiac deaths, I take it, will be all over the hospital?

MR. SCOTT: Yes.

THE COMMISSIONER: Not just in the operating room?

MR. SCOTT: They will be dominantly, I think, in the cardiology section, but they are all over the hospital.

The purple line is all other deaths, excluding cardiac deaths.

MR. SOPINKA: Where is the purple



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line?

3

MR. SCOTT: Here.

4

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THE COMMISSIONER: Well, the sum
then of cardiac and other I take it would equal
the red plus purple would equal brown; is that right?

6

7

MR. SCOTT: Plus ICU.

8

THE COMMISSIONER: The yellow line,
though, we haven't accounted for that yet.

9

10

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MR. SCOTT: What you have is all
deaths are geographic; that is to say, because they
occurred in the Hospital, they are plotted on that
line. The brown line, 7G, is geographic, too. You
are only plotted on that line if your death occurred
in the neonatal ward of whatever cause.

15

THE COMMISSIONER: That's right.

16

17

18

MR. SCOTT: All cardiac is not
geographic. That's a diagnostic line, and I will
explain how that was computed in a second. Let me go
to ICU.

19

20

21

ICU, the yellow line, is again a
geographic line in the sense that the death has to
have occurred in the ICU before the death is plotted
on this line.

22

23

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25

THE COMMISSIONER: The purple line,
which is All Others means all others of what?



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MR. SCOTT: Let me come to that in
a moment.

3

4

The blue line at the bottom is 5A
and, then, in 1980, turning into 4A and 4B, and that
again is a geographic line, you had to be on that
ward to figure in that line.

6

7

THE COMMISSIONER: All right.

8

9

Now, help me out with the purple
line then. What's that?

10

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MR. SCOTT: First of all, if you add
up all the other lines which are geographic lines;
that is, 5A, ICU, All other and 7G, you will get
the All Deaths line, the black line. In other words,
if you add up all the geographic lines, you will get
this total.

15

16

THE COMMISSIONER: All right. There
is a 5A line, and that's the green line, is it?

17

MR. SCOTT: Yes.

18

19

THE COMMISSIONER: Isn't that, the
one at the bottom, isn't that green?

20

MR. SCOTT: Aquamarine.

21

THE COMMISSIONER: I'll settle for
green.

22

MR. LAMEK: Turquoise.

23

MR. SCOTT: Trust Mr. Lamek!

24

25



1
2 THE COMMISSIONER: I just want to
3 make sure I can recognize the blue line when I see it.

4 Could you point out the blue line
5 for me, please.

6 MR. SCOTT: Here

7 THE COMMISSIONER: Oh, I thought
8 that was the green line. That is the blue line?

9 MR. SCOTT: Yes.

10 THE COMMISSIONER: That is 4A, or
11 is it 5A? It says '5A'.

12 MR. SCOTT: Well, it begins, as you
13 will recall, as Ward 5A.

14 THE COMMISSIONER: Oh, I see.

15 MR. SCOTT: And then, I think in
16 April of 1980, the 1st of April, they moved from 5A to
17 4A and 4B, and that is recorded here, just so you will
18 have a note of it.

19 THE COMMISSIONER: So, whether we
20 call it green or blue or aquamarine or anything, that
21 is all of 4A and 4B and 5A?

22 MR. SCOTT: That's right, yes. And
23 the All Other line is all other deaths, excluding
24 cardiac deaths and excluding the others noted. That
25 is, if you exclude cardiac deaths, ICU deaths, 4A-4B
deaths, you then produce the All Other line.

It certainly was so on 4A/D
as between Oct 1/79 - June 30/80
and July 1/80 - March 31/81!

- ① Check that this is so
- ② In any event, the ICU is
one of the places where
patients do. Conditionally
die.
- ③ MAY be corroboration of the
"severity cluster" hypothesis
EXCEPT that the Ward 4A/D →
ICU transfers were v. few and
therefore the jump in ICU
death rate ~~must~~ ^{may} reflect a
higher incidence of very ill
patients in OTHER areas of
the Hospital.
- ④ ? Time clustering of ICU deaths?



1
2 That is presented so you won't think,
3 Mr. Commissioner, that anywhere in this Hospital,
4 contrary to what has been reported, there is a 60 per
5 cent increase that attaches to the Hospital death
6 rate. That simply isn't so, as this chart -- a 600
7 per cent increase. That simply isn't so, as the chart
8 reveals. And, for example, we'll be dealing with
9 some particular points.

10 If you look at this chart, for
11 example, if you look at July and August of 1980, the
12 two opening months of what we call the epidemic
13 period where there is an increase of deaths in this
14 ward, you will see that there is an extravagant
15 increase of a much greater proportion in ICU deaths.
16 So that if one, in July or August of 1980, was looking
17 to find an area in the Hospital where there were an
18 escalated number of deaths, you would be attracted
19 not so much by 4A and 4B but, rather, by the ICU in
20 that case.

21 This is simply presented so that you
22 will have a complete picture and will not be obliged
23 to look only at one ward without relation to the
24 others.

25 For example also, when you come to
March, the last month of the epidemic period, where

It is helpful to do this (but not helpful
to the Hospital's position) because the chart
demonstrates that all the dramatic month-to-
month fluctuations in mortality were quite common-
place in other areas, THEY WERE NOT ON 4A/D!

Why were children not being sent from
4A/8 to the ICU?

2 possible explanations are:

(a) the ICU, because of overcrowding,
was refusing to accept patients
(as to which the Hosp. will
have to address it)

(b) 4A/8 patients were not, until
immediately prior to their deaths,
considered to be in need of
intensive care — and when,
with the sudden onset of terminal
events, the need did become
clear, the decline was too rapid
to permit transfer to the ICU
except in the case of Pacesi.



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there is an increase in 4A-4B, there, you will see
that there is a decline in the ICU rate, and we'll
have something to say about that in due course.



C
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1
2 Then in January, of course, in 1982, there is an
3 extravagant increase, the greatest ever I think in
4 history, in ICU deaths.

5 For example, I think the increase that
6 occurred in ICU deaths in 1981-1982 is far greater in
7 a gross or percentage sense than any increases that
8 occurred in this period in 4A/4B. So we wouldn't want
9 you or any members of the public to get the sense that
10 the death rate in the hospital is an evenly graded
11 exercise. It is represented by elaborate shifts which
12 are sometimes difficult to explain, and which we will
13 try and deal with, at all levels.

14 Now, as I said, I undertake in due
15 course to prove that.

16 THE COMMISSIONER: Could we also have
17 copies of it?

18 MR. SCOTT: Yes sir, we are having
19 copies made. I should also say that we will have
20 this afternoon, I thought it would be here this
21 morning, but it can't be, a bar graph over the same
22 period which I will seek to introduce which will show
23 some other factors about mortality rates in the
24 hospital.

25 EXAMINATION BY MR. SCOTT:

Q. Now, Dr. Rowe, about half-way
through your evidence, you were being asked, by Mr.

Lamek,



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whether the deaths of certain babies that occurred on -- you were asked to describe, to give your opinion as to the cause of the deaths of certain babies on 4A/4B in the epidemic period. Having done so, you were in almost every case asked, following that, by Mr. Lamek, if the death was consistent with digoxin toxicity. You gave your answer, and I think if I can summarize correctly, the upshot of most of your answers until we come down to Baby Pacsai, was that the manner of dying was consistent with digoxin toxicity in some of those cases, but there was no evidence indicative of it until the Baby Pacsai. Have I summarized that correctly?

A. Yes, I think so.

Q. And, of course, Baby Pacsai is, I think, the first baby in which there was a premortem serum digoxin level of significant elevation, would that be fair?

A. Yes. Apart from, I think, one sample in one of the earlier babies.

Q. Well, the premortem serum sample for Pacsai was taken, as I have it, on March 10th, and was 10 nanograms?

A. Yes.

Q. And I take it that while there

Because the level was > 4.7 and because
me it could not know how much
higher than 4.7, how could this
be merely "of a little concern"?

What of 3.5 ()
4.7 () ?



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had been previous ~~pre-s~~ ^{mole} ~~rum~~ samples at the three and four level, this was the first premortem sample of this kind of elevation in the epidemic. Also I am just trying to check the sample of Estrella? Estrella was 4.7, premortem?

A. I am not sure if it was

✓ greater than 4.7.

Q. Yes.

A. So it was a level about this
that was certainly of a little concern.

Q. All right, let's leave it this way then. That prior to Estrella, were there any premortem samples which gave you any particular concern?

A. No.

Q. And between Estrella and Pacsai were there any premortem samples that gave you any particular concern?

A. Can I just check that one point?

Q. Yes. I can tell you, there was Fazio 1.5; there was Floryn 2.1; Leith 2.1; Gionas 1.2; Manojlovich -- I think I have the name right, 2.2?

A. Yes.

This statement: He was asked, re
each death

- Was death, inc terminal event and then
arise & proper consistent w.
patient's clinical condition

- Also consistent w. dig in box²



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Q. And all those samples would be within, all those premortem samples would be within the normal therapeutic range?

A. Yes.

Q. So I have it then that Estrella is a cause for concern for reasons that you have given, but between Estrella and Pacsai would I have it right that there are no other cases that cause you concern with respect to digoxin toxicity?

A. No, not after examination of the levels and the time of the sampling.

THE COMMISSIONER: Excuse me, Mr. Scott, what is the number of the preliminary inquiry exhibits, it has a number I know?

MS. CRONK: It is Exhibit 32.

THE COMMISSIONER: Thank you. I am sorry.

Q. Dr. Rowe, the way your evidence in chief went, by virtue of the kind of questions you were asked, seemed to suggest that there were only two possibilities as the underlying cause of death, that is cardiac arrest of some type, or digoxin toxicity. Indeed, when I said there might be others,



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it was suggested that no one had suggested any others.
So what I want to deal with first, at some considerable
length, is what I have called as a heading "The
Mechanics of Babies Dying". Let me put a few
propositions to you, just to get started, to see if
you and I agree, and we should, because I think you
told me most of this.

First of all, the stoppage of a heart
is the characteristic final event of all deaths, is
that correct?

A. Yes.

Q. And that is cardiac arrest,
the stopping of the heart?

A. Yes.

Q. So that what happens when the
heart stops is that the muscle of the heart stops
pumping, traditionally?

A. Yes.

Q. Or characteristically, is that
right?

A. That is right.

Q. And whether the muscle of the
heart has stopped pumping or not can be monitored and
observed by an electrocardiogram?

A. You can see where there is no

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bush!



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further electrical activity of the electrocardiogram,
and the pulse and the blood pressure measurements
would tell you that it is not pumping.

Q. And I take it that the --

THE COMMISSIONER: Could I just have
that again, because there seems to be a distinction
that I haven't got.

THE WITNESS: The electrical activity,
there may be electrical activity on the electrocardio-
gram of some sort when there is still -- when there is
no action of the pump itself. So that it doesn't
necessarily mean you have to have no activity of the
electrocardiogram for the patient to be dead.

THE COMMISSIONER: But I take it, it
means something.

THE WITNESS: It is very close.

THE COMMISSIONER: So the electro-
cardiogram will show a slowing of the heart, perhaps
not a total stoppage.

THE WITNESS: Yes.

THE COMMISSIONER: Is that what you're
saying?

THE WITNESS: Yes, but you still may
have some electrical activity when the heart is not
acting as a pump. So one has to use other measures



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such as the pulse and the heart beat and the blood pressure.

Q. But it can be physically observed as well, can it not?

A. Yes.

Q. All right. Now, heart stoppage, or cardiac arrest, you have told us is the characteristic final event. I put it to you, if a man was stabbed in the back with a knife, the characteristic final event which caused his death would probably be cardiac arrest, the stoppage of his heart?

A. Yes.

Q. And that would be so if he suffered from blood poisoning in the right toe and was going to die, that cardiac arrest, or heart stoppage, would again be the characteristic final event that signified his death?

A. That would be so.

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Q. The stopping of the heart?

A. Yes.

Q. Yes. And there is ---

THE COMMISSIONER: I am sorry. I have been through some of this before. I thought that at some point the brain damage becomes so bad that even though the heart keeps going the medical fraternity consider that clinical death.

THE WITNESS: Yes. That can be ---

THE COMMISSIONER: Am I wrong?

THE WITNESS: No, that can be true too.

MR. SCOTT: But the heart is still pumping or may be still pumping.

THE COMMISSIONER: If the heart is still pumping, and I don't know, we may just be fussing about semantics.

THE WITNESS: No, I don't think it is fussing, but that is a special category. I think that is different from most situations.

MR. SCOTT: The reason I am concerned about this, Mr. Commissioner, is we have thought about cardiac arrest as the cause. That is to say the underlying cause of death. In fact it may be the underlying cause but there may be, and I have a list



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of 14 other underlying causes which lead to cardiac arrest properly understood; that is the final stoppage of the pump action.

THE COMMISSIONER: Yes. If I understand the various theories on this, the causes of death that have been advanced so far are heart disease and digoxin poisoning.

MR. SCOTT: There are 13 more.

THE COMMISSIONER: The symptoms of both, I suppose, could be cardiac arrest.

MR. SCOTT: Well, we will be coming to that.

THE COMMISSIONER: Yes. Well, at least not the symptoms so much as the demonstration. But the cause of death I don't think anybody has said has been cardiac arrest.

MR. SCOTT: Well, I ---

THE COMMISSIONER: Or at least I haven't heard that.

MR. SCOTT: Well, perhaps if I can ask you just this once, Mr. Commissioner, to be patient.

THE COMMISSIONER: Yes.

MR. SCOTT: Around about November you can lay into me seriously and tell me to stop,



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but I would really like to deal with ---

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THE COMMISSIONER: The only reason

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I am doing that is not to demonstrate anything but

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my own ignorance. When I don't understand, that is

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when I ask the question.

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MR. SCOTT: I am very grateful

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for those interventions.

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Q. Now, Dr. Rowe, do I understand

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from the evidence you have previously given that

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there are essentially two types of heart stoppage?

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The first is when the heart simply stops pumping;

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that is when there is no contraction at all in the
muscle.

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A. Yes.

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Q. Yes. And the second is

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where the contraction continues but is ineffective
to pump sufficient blood out of the heart.

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A. Yes. In ventricular fibrilla-

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tion.

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Q. Yes. So that is ---

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THE COMMISSIONER: That is what is

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called ventricular fibrillation?

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THE WITNESS: Fibrillation, yes.

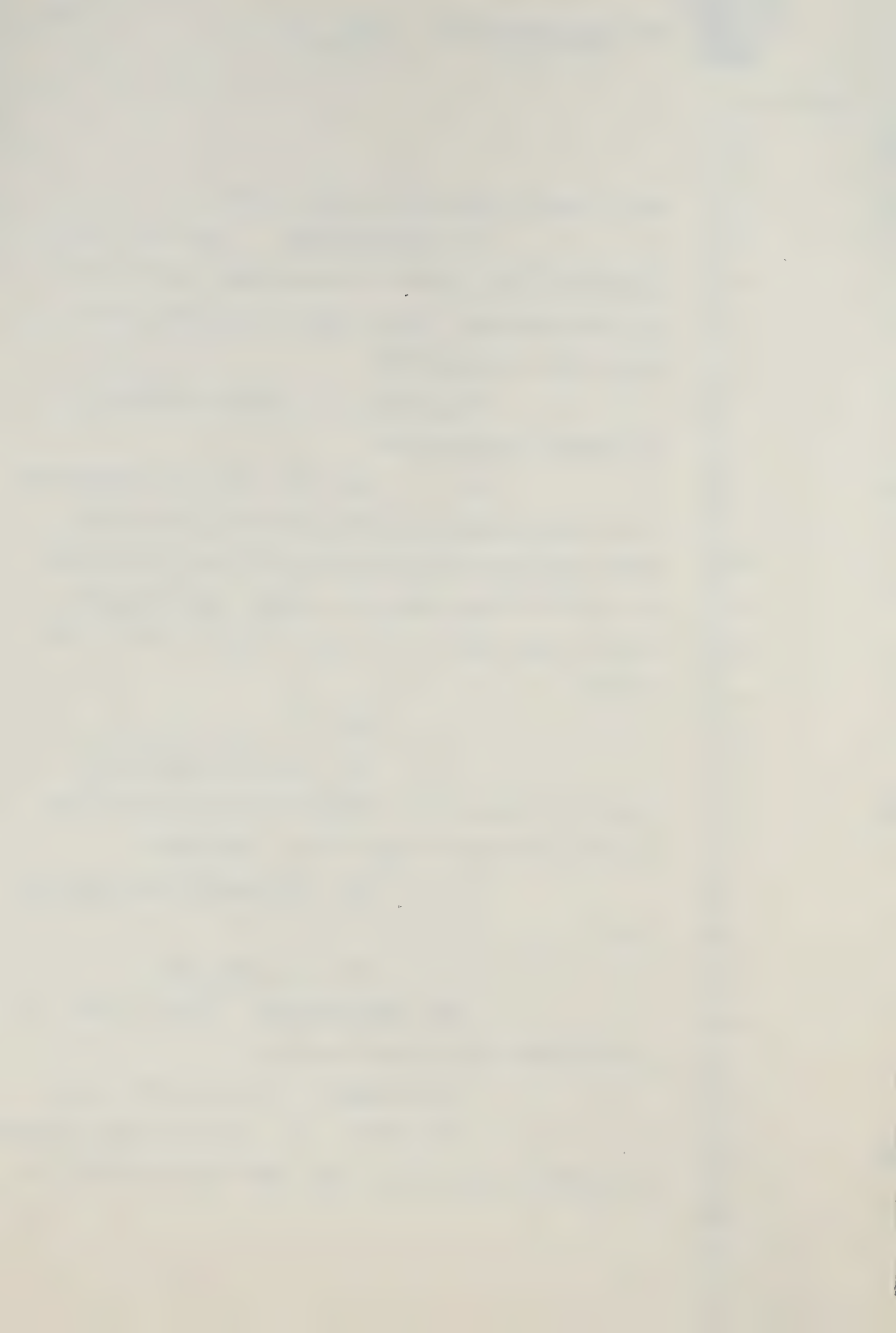
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MR. SCOTT: Q. So the heart stoppage

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or cardiac arrest as a final event causing death is

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typical of two types: one in which the muscle contraction ceases altogether, and the second in which the muscle contraction continues but is not sufficiently powerful to pump blood out of the heart in sufficient quantities?

A. Yes. And eventually that type becomes an arrest too.

Q. Yes. And what happens in the fibrillation case is that there is a loss of oxygenation in the other parts of the body? They have no nourishment and those body parts die.

A. Yes.

Q. Now one other term that we have dealt with and perhaps it is obvious, but resuscitation, and tell me if I am correct about this, is an effort by physical means, drugs or perhaps otherwise to get effective contractions underway so that they are self-sustaining?

A. Yes, that is true.

Q. So when you have a heart stoppage or a cardiac arrest which would be the final event, unattended, of all deaths, resuscitation is introduced in appropriate cases to mechanically or chemically or by some other means get the contractions to begin again?



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A. Yes, that is the intent, yes.

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Q. Now when that fails and the

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heart can't be induced to pump again, the patient is
5 dead?

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A. Yes.

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Q. And when the patient dies a

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question asked is what caused the patient to die?

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That is the layman's question, isn't it?

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A. Yes, it is.

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Q. And is that for a doctor

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the same question as why did the heart contractions
or effective heart contractions stop?

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A. Yes. That would be the

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question we would ask.

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Q. So when you have a cardiac

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arrest you don't assign cardiac arrest as the cause

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of death; you (though it is technically the last

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moment) you ask what caused that cardiac arrest?

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A. Yes.

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Q. Am I right?

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A. You are right.

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Q. And I take it that that

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observation that the critical question, "What caused
the cardiac arrest by which we will all die? What
caused it?" is as appropriate for babies as it is

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for adults?

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A. Yes, it is.

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Q. Now having said that, I want

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to take you to some of the ways cardiac arrest can

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be caused, and I want you to think in this instance

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particularly of babies and particularly in this

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Hospital and the so called epidemic period.

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I want to see if I can't get, with your help, a list of the possible causes that led heart contractions to stop or caused heart cardiac arrest typically in this Hospital, and indeed at the end I may ask you to give us (I don't think you have prepared it yet, but you are going to be around for a while) a list of the 36 babies assigning a cause to the cardiac arrest which led in most cases to their death. In all cases to their death.

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Well let's deal first of all with heart failure. Is heart failure a cause or a potential cause of cardiac arrest?

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A. Yes, it is.

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Q. And what does a doctor mean

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when he talks about heart failure?

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A. Well, he means that the state of the heart contractions have reached a point where they cannot adequately pump blood around the body.

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Q. Yes. And is that characteristically a phenomenon in which the muscle of the heart tires out and stops?

A. That is the simplest way of putting it.

Q. Yes. And that tiring out of the muscle of the heart in heart failure, is that characteristically a result of stress or pressure created by abnormal construction of the heart?

A. Usually - it may occur from infection of the heart muscle, but it is something that affects the performance of the muscle cells of the heart, yes.

Q. All right. Now two weeks ago when we had the charts of the various babies on the lectern, you led us through some babies who had hearts that were abnormal in physical structure.

A. Yes.

Q. And indeed I think most of the babies in your Ward 4A and 4B in the epidemic period did, did they not?

A. I think there were only three who did not have abnormal hearts.

Q. All right. Now apart from anything else is a baby with a heart structure which



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is abnormal or defective a candidate for heart failure?

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A. Particularly - that is true particularly if the malformation is not a very mild one.

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Q. All right.

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A. Unless it is mild they are candidates for failure.

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Q. So that that baby with an abnormally structured heart will die like all babies do because of cardiac arrest, but one of the potential causes for the cardiac arrest is heart failure, characteristically, the abnormal structure of the heart and the fact that the muscle tires under the stress of that abnormality?

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A. Yes. The mode may be a little different for different malformations, but heart failure in the end is a good general term for what takes place in that situation.

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THE COMMISSIONER: Could I interrupt for just a moment? What are the external symptoms of the difference between the ventricular fibrillation and the true stoppage of the heart?

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THE WITNESS: There is no external difference. The only way you can tell that is by the

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electrocardiogram. Unless the chest were open and you were actually looking at the heart in the gross form.

THE COMMISSIONER: When in these medical records they refer to ventricular fibrillation as being observed, they can't be sure?

THE WITNESS: They can be sure if they see the electrocardiogram but not otherwise. The electrocardiogram is specific for ventricular fibrillation.

THE COMMISSIONER: I see. All right. So it will show some ---

THE WITNESS: It will show a very definite and consistent characteristic rhythm abnormality.

MR. SCOTT: Q. So that a baby that has a grossly deformed septum, for example, an abnormal structure of the heart, may be a candidate for heart failure strictly speaking?

A. Yes.

Q. That is the mechanical stress on that defective organism may lead simply to its wearing out?

A. Yes.

Q. Now at this point I want to



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stop because in every one of these deaths - I shouldn't say every one but in close to every one Mr. Lamak, having got you to give your opinion as to the death, sought to invite you to compare the death of Baby X or Baby Y in terms of the presence of bradycardia, vomiting, the sudden deterioration or onset, ventricular fibrillation, arrhythmia and shallow respiration. And you may recall his point was, well, Baby X appeared to be stable and then these things happened, and that distinguishes that death from the others.

Do you remember the line of questioning that he put to you in each case?

A. Yes.

Q. Yes. All right. Now I want to ask you about those.

Dealing with the first cause of heart stoppage, heart failure, and before I do I would like to read you evidence given by Dr. Ralph Kauffman at an inquest into the death of a baby, Garry Murphy, who died outside the epidemic period and after the epidemic period and whose serum revealed digoxin toxicity. Readings beyond the therapeutic range.

Now, first of all, do you know who Dr. Kauffman is?



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A. Yes, I do.

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Q. And I take it he is a well

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known pharmacologist?

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A. Yes, he is.

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Q. In Chicago?

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A. Yes. Detroit.

8

Q. Detroit?

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A. Yes.

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Q. And he was asked and he was

11

called I can tell you by the Crown in this case

and he was asked ---

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THE COMMISSIONER: Was this the

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inquest?

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MR. SCOTT: This is the inquest.

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Q. He was asked about the signs
of digoxin toxicity.

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MR. PERCIVAL: What page is that?

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MR. SCOTT: It is page number 12 of
the Kauffman excerpt. I don't think it is page 12
of the entire transcript.

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Q. I just want you to listen to
the question and answer and then I'm going to ask
you if you agree with it or have any comment to make
about it.

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"Q. The signs of toxicity,

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"especially in an infant, if you had vomiting which we have heard from a previous witness, Dr. Hesslein, poor feeding, irritability, are these necessarily things which would hold up a red flag to you and say ah-ha, there is digoxin toxicity present?"

The doctor says:

"I have to respond to that by saying that the symptomatic signs of digoxin toxicity in infants are rather non-specific, and usually are symptoms that can be due to other factors, and it is difficult in many situations in a clinical situation to be certain whether or not a specific symptom is due to or not due to digoxin in a child. And this is where levels come in handy sometimes to help you sort that out. Vomiting, it is true that vomiting, loss of appetite, irritability, can be symptoms associated with toxic digoxin effects. They can also be associated with a myriad of other things in infants this age, and that is why it is so difficult to make a definite association."



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Do you agree with that statement?

A. Yes, I do. I don't think there would be any difficulty in any pediatric cardiologist agreeing with that statement.

Q. All right. So that, when Mr. Lamek asks you, in the case of the death of Baby X, well, wasn't there vomiting here, is it possible that vomiting, as Dr. Kauffman says, can be attributable to digoxin toxicity as well as a myriad of other causes?

A. Yes.

Q. All right.

A. We have examples of babies who vomited in this group where we know that the digoxin levels were absolutely within the normal range.

Q. All right.

Let's take then -- we are dealing with the first cause of heart stoppage, which is heart failure, a functional abnormality in the heart, and I want to go through the things that Mr. Lamek asked you about.

When you have a case of heart failure, you will typically have an abnormality of the heart structure?

A. An abnormality or an infection.

Q. Of the heart structure?



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A. Of the heart structure, yes.

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Q. Yes. Is it accompanied by

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bradycardia from time to time?

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A. It may be.

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Q. Yes. Is it accompanied, as

death approaches, with vomiting from time to time?

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A. Yes, especially if the failure

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is bad.

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Q. Yes. Is the terminal event

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often a sudden onset?

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A. Yes.

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Q. Is it accompanied by ventricular

fibrillation from time to time?

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A. Yes. There is a proportion

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that do that.

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Q. Yes. Is it accompanied by

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arrhythmia?

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THE COMMISSIONER: I'm sorry, let us

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pause there for a moment.

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Is ventricular fibrillation, is that

a progress towards the stoppage of the heart?

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THE WITNESS: Yes. It is a mode of

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the heart stoppage, but it is not necessarily present

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in every baby who dies.

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THE COMMISSIONER: Well, I am still

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2 having a little trouble in distinguishing between them.

3 Obviously, the heart stoppage, I can
4 understand with no difficulty at all, but ventricular
5 fibrillation, I am having some problems. I know the
6 result of it is you don't get enough blood around the
7 rest of the body.

8 THE WITNESS: Yes.

9 THE COMMISSIONER: But what does it
10 mean with the heart? What's happening?

11 THE WITNESS: Well, it's an end stage
12 situation; the heart is just --

13 THE COMMISSIONER: Barely beating.

14 THE WITNESS: Has a chaotic obstruc-
15 tion. I think it has been referred to in previous
16 statements by pharmacologists in some of these hearings
17 I am not sure whether it is the inquest or not - that
18 the heart looks like you had a bag of worms in your
19 hand or you had a handful of worms and you see very
20 chaotic but ineffective contraction of muscle. That
21 is just one way in which the heart may stop. The
22 usual way in babies is that the heart just simply
23 slows and then stops. But in a proportion which, in
24 this particular group of patients, was somewhere around
25 25 per cent, ventricular fibrillation may be the
mechanism.



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MR. SCOTT: Q. Well, I want to take you back. You told us that heart stoppage, the final event, that could be caused two ways, typically.

A. Yes.

Q. One, by the heart slowing down and stopping or simply stopping.

A. Yes.

Q. It doesn't necessarily have to slow down, does it? It may just stop.

A. Yes. But most times, it does slow down.

Q. All right.

THE COMMISSIONER: I think that it would have to slow down even if it is almost immeasurable, but it would have to slow down somewhat before it stopped, I would think, does it not? I'm not sure that --

MR. SCOTT: Why?

THE COMMISSIONER: Well, I don't see how, when you're going and you stop, you don't slow down.

MR. SCOTT: Well, a pump that is pumping at a given rate can stop instantly, it simply stops the next pump; it doesn't have the next pump.



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But no matter.

THE COMMISSIONER: Well, I suppose it can. I don't imagine though that is what happened, but perhaps I'm wrong. But that isn't usual, is it?

THE WITNESS: Most often there is bradycardia or slowing first.

MR. SCOTT: All right.

THE COMMISSIONER: Yes.

MR. SCOTT: Q. The second method that you were telling us about is fibrillation, the circumstance in which the contractions continue to occur but are chaotic. It isn't necessarily that they are weaker, is it, though they may be?

A. They are weaker and they are completely disorganized so that there is no uniformity to the function at all.

Q. All right. And the result of fibrillation, that chaos in the pumping, is insufficient blood and therefore oxygen doesn't get out to the system?

A. There is no blood discharged to the system.

Q. Yes. Well now, I asked you - we were going through Mr. Lamek's list - is ventricular



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fibrillation found from time to time in cases of heart failure which causes cardiac arrest?

A. Yes.

Q. Yes. Now, how about arrhythmia, is it found from time to time in heart failure which gives rise to cardiac -- the ultimate stoppage of the heart?

A. Yes.

Q. What about shallow respiration?

A. That, too.

Q. Yes. So, all those factors which Mr. Lamek emphasized, if I read his examination right, can be found in cases of heart failure, which is, on our list, the first cause of cardiac arrest?

A. Yes.

Q. Yes.

Well now, let's come to a second cause of cardiac arrest - hypoxia.

I wonder if you could tell the Commissioner what hypoxia is.

A. Hypoxia is a lack of oxygen. It is not complete absence of oxygen but it is a considerable and important lack of oxygen available to the systems of the body.



Rowe

ex. (Scott)

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Q. May that lack of oxygen occur in either the heart muscle or the brain?

A. Yes. In fact, if anybody is hypoxic, it affects all organs but it particularly affects those which need to have oxygen particularly strongly for their effective action, like the brain and the heart.

Q. And what is the consequence of this lack of oxygen in the brain or heart muscle?

A. Well, the lack of heart oxygen affects the cellular function of the organ. It does not allow the organ to produce energy in the usual way; it interferes with the membranes of the organ and of the cells of the organ and, so, has that sort of effect.

Q. Does it have any affect on the contractions or the pumping that the heart does?

A. Yes, it can do. Now, the heart is fairly tolerant of hypoxia in babies.

Q. Yes.

A. So that you can have low oxygen, a lowered oxygen in the system without necessarily causing any immediate problem in terms of slowing of the heart. But when other events are added to it, such as acidosis or anything that would increase the metabolic demands - the actual requirements of the



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ex. (Scott)

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body, such as fever or irritability or something like that - that can tip the scale and, then, the effect of hypoxia can completely slide over the scale.

Q. I just want to be sure I understand its effect. If you have hypoxia at an extreme level --

A. Yes.

Q. -- which is the shortage of oxygen in the brain or in the heart muscle, do I understand you to say that what that does is reduce the effectiveness of the heart muscle as a muscle?

A. It can.

Q. Yes. And what effect does that have in an extreme case on contractions?

A. Well, it will reduce contractions.

Q. Yes. To what point?

A. To the point where it can stop.

Q. All right.

Now, I take it that hypoxia can stop heart contractions even in the case of a perfectly normal heart.

A. Yes. We're not quite sure, in that situation, whether that effect is direct on the



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heart muscle or whether it is a brain effect.

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Q. All right. Well, let me take the case of a baby in the hospital who has a deformed heart. That baby is at risk - I am not asking you to quantify the risk - is at risk theoretically because of the first cause of death, heart failure, stress created by a deformed heart, but may also theoretically be at risk if hypoxia sets in?

A. Yes.

Q. And in both cases the terminal event will be a cardiac arrest or the stopping of the heart?

A. It could be.

Q. Yes. One will -- for one, the first, the cause will be the abnormality of the heart; in the second theoretical case, the cause will be hypoxia?

A. Yes.

THE COMMISSIONER: It could be both, though?

THE WITNESS: It could be both.

MR. SCOTT: Exactly.

THE WITNESS: It can have many combinations.



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Q. The more options there are for causing the stoppage of the heart, Dr. Rowe, I suggest to you, the more difficult it is to determine what caused the heart to stop?

A. Yes, that's true.

Q. Because the range of the possibilities gets greater, am I right?

A. Yes.

Q. All right.

Now, let's talk about hypoxia. Were there babies in this ward who you think may have suffered from hypoxia as they approached their deaths?

A. Yes.

Q. All right. Now, I am going to ask you at the end to make, when you have some free time, a list - I haven't asked you to do this - of the babies that you think would be in that category.

THE COMMISSIONER: But before we get on with that, I take it that you can suffer from hypoxia without any disease of the heart; is that possible?

THE WITNESS: Yes, you can. That is usually from disease of the lung.

MR. SCOTT: Q. Well now, let me



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just go at hypoxia again and take Mr. Lamek's list.

If you had a death that was being caused by hypoxia where there was no structural defect; in other words, I'm talking theoretically - take the pure hypoxia death.

Are you with me so far?

A. Yes.

Q. Might that be accompanied by bradycardia?

A. It is.

Q. It is?

A. Yes.

Q. Might it be accompanied by vomiting?

A. Yes.

Q. Might it be accompanied by sudden deterioration or onset?

A. Yes.

Q. Might it be accompanied by ventricular fibrillation?

A. Yes.

Q. Might it be accompanied by arrhythmia?

A. Yes.

Q. Might it be accompanied by



E12

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shallow respiration?

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A. Yes.

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Q. Yes. Might it be accompanied

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by seizures?

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A. Yes.

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Q. So, when you have hypoxia,

8

you may exhibit all or some or any combination of

9

those symptoms as you approach the climactic moment
of death?

10

A. That's true.

11

Q. All right.

12

Well now, that's hypoxia, and I will

13

be asking you in the end for a list, but are you aware

14

now of any babies in the epidemic period in 4A and 4B

15

who had both heart abnormalities and were, therefore,

16

susceptible to heart failure and the symptoms and
signs of hypoxia?

17

A. Yes.

18

Q. Now, the third, are you

19

familiar with the term "sepsis"?

20

A. Sepsis, yes.

21

Q. Now, is sepsis itself a

22

potential independent cause of heart stoppage?

23

A. Yes.

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Q. Yes. Can it exist without

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any malformation in the heart at all?

A. Yes, it can.

Q. Yes. Now, would you tell the Commissioner and me what sepsis is.

A. Well, sepsis is a term that is used, that I suppose would best say that an individual is septic; that is, that he has a --

Q. At the Municipal Board, that has a specific connotation; so, I would be careful about using it.

A. He has a bloodstream infection.

Q. Is that a bacterial infection?

A. With bacteria, usually, yes.

Q. And when a baby has that bloodstream infection --

THE COMMISSIONER: Is this demonstrated by pus?

THE WITNESS: No. It may not be demonstrated by pus at all.

THE COMMISSIONER: How? What?

THE WITNESS: The only way that you can tell -- it may be demonstrated by pus at some stage, but in many babies, particularly where they - and this is the group we are really, I think, looking at mostly, in the patients that are under consideration



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in this hearing, the babies are young and the speed with which sepsis can cause death in the very young babies is extraordinarily rapid; so that there may be no time for pus to appear visibly anywhere. So, the only way that this can be recognized is by the overwhelming nature and rapidity of the illness and the finding of bacteria in the bloodstream.

MR. SCOTT: Q. Well, just to be sure, Dr. Rowe, that I have it, I take it sepsis -- would you call it a disease?

A. Yes, it is a disease.

Q. All right. And it is a disease that might exist entirely apart from any abnormality of the heart?

A. Oh, yes.



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/DM/ak

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Q. But may exist with an abnormality of the heart?

4

A. Yes.

5

6

Q. But they are unconnected as a matter of theory?

7

A. Yes.

8

9

Q. And when you have a blood stream infection that is called sepsis, what does that do to make your heart stop?

10

11

A. Well it produces a toxic effect on the heart, the toxins from the bacteria.

12

Q. In the blood?

13

14

A. In the blood. It affects the heart function by a direct toxic effect, especially in sepsis in small babies.

15

16

17

18

Q. Right. And the toxic effect I take it is the infected blood going to the heart and the infection being transmitted to the heart muscle?

19

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A. The toxins from the bacteria, the release of materials from bacteria which is toxic, the cells.

22

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Q. Does that affect the heart muscle cells?

24

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A. Yes, it affects every cell.



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Q. And what may the result be if that is uncontrolled?

A. Death.

Q. Well, how is the death caused? You will have to move with me at kindergarten level. You say death, when the toxin has travelled to the heart and infected the heart muscle what effect classically might it have on contractions in the heart?

A. The end result is the failure of the muscle to contract, but the method of getting there is that the toxin affects the membranes of the heart, affects the energy arrangements within the heart and so it disrupts the whole biochemical function of the heart.

Q. But is the result in the end that the contractions of the heart stop's?

A. Yes.

Q. And do you then have a heart stoppage or a cardiac arrest?

A. Yes.

Q. And that cardiac arrest may, in the use of layman's terms, be caused by blood poisoning that has no relation to a defective heart.

A. That is correct.



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Q. And where there cases of that in the epidemic period in Wards 4A/4B, or cases that suggested that as a cause?

A. There were cases that suggested, I am not sure of the total, but blood stream infection is something that everybody thinks about immediately a baby is seen to be ill because of the fact that unless you act on it right away then you lose the opportunity to save the baby. There will be, I think, many instances in that series of patients, in whom action was taken by physicians because of a real concern that sepsis might be operating, not always sustained by the subsequent analysis, but they had to act with antibiotics and supportive measures immediately.

Q. So if you have sepsis in a baby with a normal heart, is it dangerous?

A. Yes, it is very dangerous.

Q. And can lead to death?

A. Yes.

Q. If you have sepsis in a baby with a grossly deformed heart, is the danger any different?

A. It is higher.

Q. Why?



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A. Because the heart is already -
if the heart is abnormal and not functioning well,
normally because of its malformation, then if you
load it with toxic effect you can make things that
much worse. On top of that the sepsis produces a
huge increase in the metabolic demand on the heart,
that is this fever, the heart is beating faster and
you put a new load as well as a toxic effect of the
bacteria on the heart. So it is a combination that
is pretty lethal.

Q. So the sepsis not only poisons
the heart muscle, but the presence of the sepsis
leads the heart to be obligated to contract harder
and faster?

A. That is correct.

Q. To offset the sepsis?

A. Yes.

Q. So wouldn't the new word,
people call that a synergistic effect?

A. Yes.

Q. I have wanted to use that
word for some time.

MR. PERCIVAL: How do you spell that?

MR. SCOTT: I haven't the faintest
idea.



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Q. To make it just clearer, if you have a baby with a congenitally abnormal heart who has both hypoxia and sepsis, is the risk of heart stoppage increasing?

A. Yes.

Q. And are you ever able to say with any finality which of the mechanisms was the dominant mechanism to cause death?

A. It is sometimes very difficult to do that.

Q. Now let us deal with sepsis. You have told us that it may occur even when there is a perfectly normal heart, and I take it it does in your experience?

A. Yes.

Q. May it be accompanied by bradycardia?

A. Yes.

Q. May it be accompanied by vomiting?

A. Yes.

Q. May it occur suddenly, or a deterioration occur quickly?

A. Oh, yes. We have had examples in the neonatal period where babies have been admitted



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and while they are being examined, having been thought to be at least only moderately ill, just suddenly die.

Q. In moments?

A. In moments.

Q. You see the trouble with this is that as I approach my final event as we seem to be calling it here, I anticipate that I will be allowed a leisurely period when I will move in and out of hospital suffering from heart failure and be allowed to slide gradually off the precipice, that is how we like to think about these things. Does that happen in the case of babies?

A. It can happen but it is much more common that they will deteriorate more rapidly than that.

Q. And why is that, I'm thinking of something else now, but why is that?

A. Well, it is a combination of events. There are a whole host of things that occur. Babies can become acidotic very quickly so that they have the additive effect of acidosis. They can stop breathing, especially if they are small. I am talking about small babies which is really what we are talking about in general. If they have had heart disease that has affected them for some weeks



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2 or months, then they have usually failed to thrive,
3 so they have fewer energy stores, they are not as able
4 to respond to the demands that are put upon them if
5 they have any additional infection, or a problem of
6 that sort.

7 Q. I have taken you off course
8 here for a moment. I just want to be sure I have it.
9 We have dealt so far with three causes of heart
10 stoppage: heart failure; hypoxia; and sepsis. Do
11 I have it right that all the indicia that may
12 accompany digoxin toxicity may be found in the manner
13 of babies dying from those totally unrelated disorders?

14 A. Yes.

15 THE COMMISSIONER: I'm sure that is
16 right, but you stopped at the end, half way through
17 with sepsis.

18 MR. SCOTT: Oh, did I?

19 THE COMMISSIONER: Yes. Does it
20 also include ventricular fibrillation, arrhythmia,
21 shallow respiration and seizures, or does it
22 encompass those?

23 THE WITNESS: Yes.

24 THE COMMISSIONER: Does it exhibit
25 those things?

THE WITNESS: It can. It depends



1
2 upon the age a bit as to what proportion of babies
3 with normal hearts that will have ventricular
4 fibrillation. It is unusual for babies with normal
5 hearts to have ventricular fibrillation as a means
6 of stoppage, but it is possible for them to do that.

7 Q. I take it it is not a matter
8 of concern here, because with three exceptions all
9 the babies had abnormal hearts of various degrees
10 of severity?

11 A. Yes.

12 Q. Well now, number 4, "Respiratory
13 Illness".

14 MR. PERCIVAL: Mr. Commissioner,
15 what about the other three? We talked about
16 fibrillation, what about the other three?

17 MR. SCOTT: The other three modes
18 of dying that I referred to ---

19 MR. PERCIVAL: I'm talking about
20 the arrhythmia and shallow respirations and the
21 seizures.

22 THE COMMISSIONER: You indicated
23 that those may be evident.

24 THE WITNESS: Yes.

25 THE COMMISSIONER: As sepsis?

THE WITNESS: Yes, indeed.



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MR. PERCIVAL: Thank you,
Mr. Commissioner, I didn't hear it. Thank you.

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MR. SCOTT: Q. I think you have told
us that there are cases that suggested the septic
problem in this epidemic period?

7

A. Yes.

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Q. Now, number 4 "Respiratory
Illness". Will you tell us what that is and what
impact, if any, it may have on contractions in the
heart?

11

12

13

A. Well, the respiratory disorders
are of a large, and different number of specific
types, but the most ---

14

Q. May I interrupt you, Doctor?

15

A. Yes.

16

Q. What do you mean when you say
a respiratory disorder?

17

18

A. Anything wrong with the lungs
or the airways to the lungs.

19

Q. All right.

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A. So you may have disturbances
that interfere with not the lung itself but with the
way in which air gets to the lungs, obstructs the
airway. In a small baby it is very important because
small babies have difficulty if they have minor



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2 obstructions of the nasal passages, they can very
3 nearly asphyxiate if that happens on occasion,
4 because they are not very good at breathing through
5 the mouth.

6 Q. Or blowing their nose?

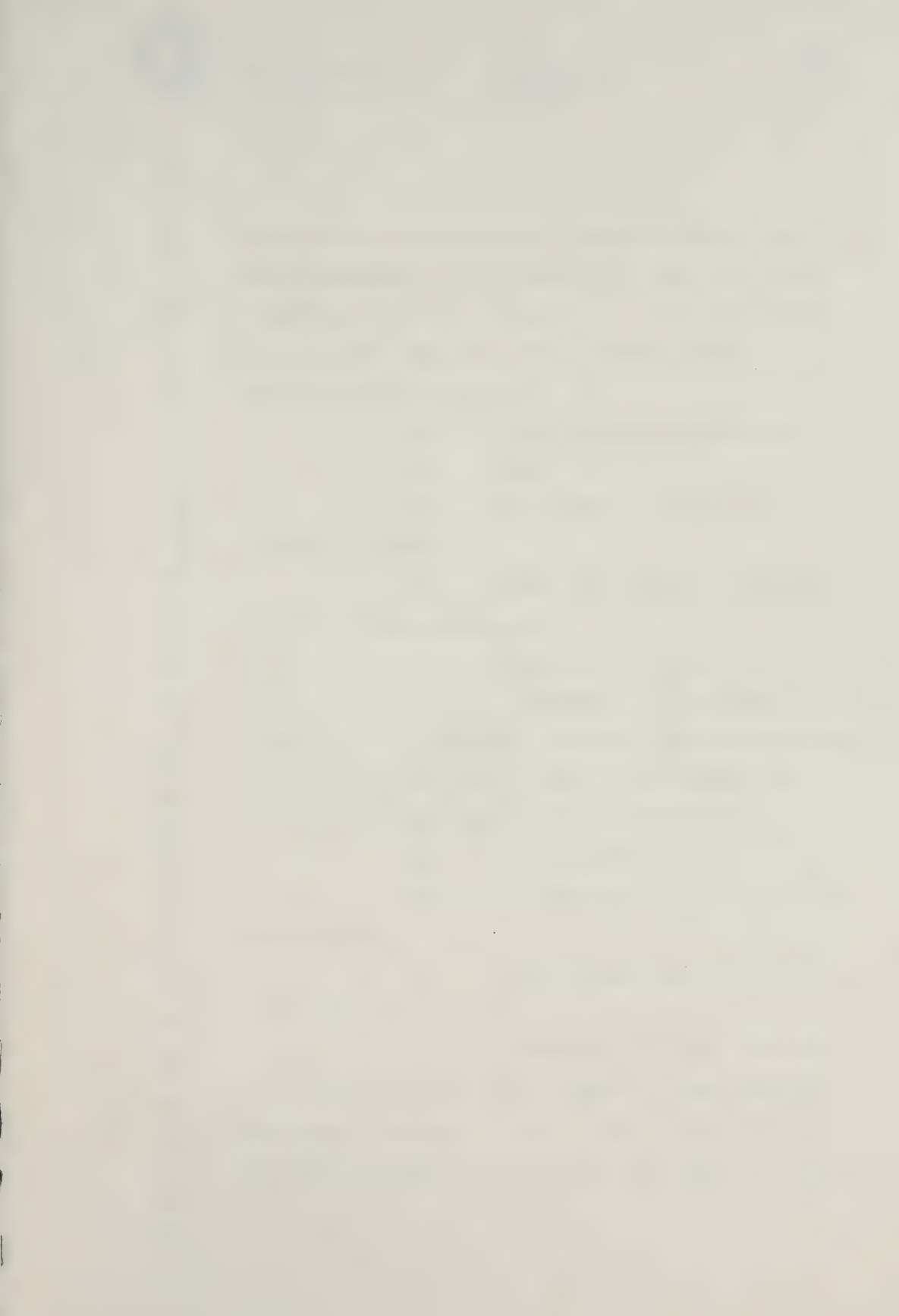
7 A. That's right. In fact there
8 are a number of conditions described in young
9 children where airways obstruction can produce
10 very severe and fatal consequences. Even having
11 tonsils that are too big and meet in the middle,
12 like kissing tonsils, may obstruct the airways in
children to the point where can die.

13 Q. I take it what that kind of
14 obstruction does, it is a respiratory ailment or
15 illness that prevents air from getting to the lungs.

16 A. Air getting in and carbon
dioxide getting out.

17 Q. And if that happens what is
18 the effect on heart contractions?

19 A. Well, if you have an obstructive
20 airway like that there is tremendous respiratory
21 effort, that is this tugging and heaving to try and
22 get air in and out. That can produce enormous
23 fluctuations in the - pressures that are inside the
24 chest. Normally there is a negative pressure inside
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the chest, but when you are struggling to get air in the effort that can be induced by a baby is of such a magnitude that you can produce a swing in pressure that is about the level of your blood pressure, an enormous swing of pressure.

7

Q. That is airway obstruction?

8

A. Yes.

9

10

Q. As a kind of respiratory illness are there?

11

A. Well, you can have pneumonia, that is a fairly common one.

12

Q. What is it I mean?

13

14

A. Pneumonia is an inflammatory condition of the lungs at the sac level of the alveolar sacs which is the distal part of the lungs, the part where the exchange of air is taking place.

15

16

Q. Is it viral?

17

18

A. It may be viral, or it may be bacterial.

19

20

Q. What is the effect of that infection?

21

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23

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A. The effect of that infection is to interfere with the exchange of gases, oxygen and carbon dioxide. It has a toxic effect if it is - from the bacteria as well. Those are the main causes.



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Q. Can it lead to a stoppage of
contractions?

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A. Yes.

5

Q. Of the heart muscle?

6

A. Yes, it can.

7

Q. And cardiac arrest?

8

A. Yes, it can, and especially
this is true in small babies.

9

Q. Now, what about atelectasis?

10

A. Atelectasis is a collapse of
a part, or segment, or part of the lung.

11

12

THE COMMISSIONER: Can we have that
spelled?

13

14

THE WITNESS: A-t-e-l-e-c-t-a-s-i-s.

15

THE COMMISSIONER: Yes, thank you.

16

MR. SCOTT: Q. What is atelectasis?

17

A. Atelectasis is a collapse of
a portion, or a total collapse, meaning the whole of
one lung or both lungs.

18

19

Q. And is that, generally
speaking, a respiratory illness of the type we are
talking about?

20

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A. Yes, regardless of respiratory
illness.

22

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Q. What is the consequence of
that?

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A. Well, it depends on the severity. It is fairly common in ^many babies who have segmental or collapse of part of a lobe, not to be necessarily seriously affected by that. If it becomes extensive, or if it involves large amounts of smaller segments of lung then it can interfere with the oxygen exchange and carbon dioxide release.

Q. What is lung congestion, is that a respiratory illness?

A. Lung congestion is when the lung is full of blood, and that is usually because of the presence of heart failure. So that in most patients who have heart failure there is a degree of interference with lung function. Sometimes in small babies the actual size of the heart when it is deformed, and malformed and there is heart failure, can be such that it will compress the lung and so cause collapse.



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Q. Well, under the general heading
respiratory illness, we have dealt with four different
kinds of respiratory illness. Are there others as
well?

A. Oh, there are probably others,
but that is the major group, I would think.

Q. All right. And can each of
those lead, if severe enough, to stoppage of heart
contractions and cardiac arrest?

A. Yes, they can.

Q. And can that occur even in the
case of a normal heart?

A. Yes.

Q. Now, add a respiratory illness
to a case of an abnormal heart, and what do you get?

A. You have an additive effect.

Q. Yes. And were there cases in
this epidemic period where babies died and where
there is evidence pointing to respiratory illness --

A. Yes.

Q. -- as one of the causes?

A. Yes, there were.

Q. Let me take you through the
list again. Is death -- that is heart stoppage --
caused by respiratory illness, commonly accompanied by

If the only time he has seen vent. fib^l when respiratory problems are the major cause of death is one of the patients here [find out which one] is it not arguable that the occurrence of vent. fib^l points away from resp^y. problem as cause and is dig into^x of which fib^l is known to be a symptom?



G2

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bradycardia?

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A. Yes.

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Q. May it be accompanied commonly

5

with vomiting?

6

A. Yes.

7

Q. May it be accompanied -- does

8

it exhibit a sudden outset or a sudden deterioration

9

in the patient?

10

A. Yes.

11

Q. May it be accompanied by

12

ventricular fibrillation?

13

A. I don't know. I haven't seen
that, but it may. *Has know if haven't seen?*

14

Q. All right. May it be

15

accompanied -- I only want you to tell us --

16

A. Yes, we do, in fact, have one

17

patient --

18

Q. -- you are quite right, if you
have seen it or have read about it.

19

A. We have one case in this series.

20

I have forgotten one case.

21

Q. This series you are talking

22

about?

23

A. Yes.

24

Q. May it be accompanied by

25



G3

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arrythmia?

3

A. Yes.

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Q. May it be accompanied by

5

shallow respiration?

6

A. Yes.

7

Q. May it be accompanied by

8

seizures?

9

A. Yes.

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THE COMMISSIONER: At some point,
Mr. Scott, whenever it is convenient...

11

MR. SCOTT: Now.

12

13

THE COMMISSIONER: I'm not too sure
how the logistics are going to work out, so we will
make it 20 minutes and you can tell me if you don't
need that another time.

14

15

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We will take 20 minutes.

17

---Short Recess.

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MR. SCOTT: Mr. Commissioner, if you
will permit me, I am going to take another crack at
this chart.

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THE COMMISSIONER: Yes. Just before
you do, I want to, just for the comfort and solace of
the inner man or woman, we have -- Mr. Diplock of
the Municipal Board was concerned about the comfort of
Counsel, and they have arranged... There is a lounge



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on the 19th Floor, which has a very simple-minded -- it is where the Assessment people -- the people have taken over from the County Court in the Assessment Appeal, where they are there. They are not working today apparently but they have a lounge. It is available for Counsel.

There is a combination which is a very difficult combination to work. If you forget the combination, you use a credit card and you can get in very easily. Mr. Lamek says that we can give you some supplies or something like that and we just hope that you behave and don't litter the place because when they do come back and get working again, they may take objection if there is too much noise. But they are really trying to make you comfortable. So if you want to use that for coffee purposes at the breaks, just do that.

It takes a little time to get up there. There is the little complication about getting in, but that is it.

MR. LAMEK: Room 1900.

THE COMMISSIONER: 1900, yes.

MR. LAMEK: And I suggest not to be used until tomorrow, and we can get some supplies up there.



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THE COMMISSIONER: Yes. All right.

MR. LAMEK: It is on the other bank
of elevators.

THE COMMISSIONER: Now, Mr. Scott, do
you want to take over?

MR. SCOTT: Yes. It may be that I
was not clear when explaining one matter on this
chart and that I failed to disclose another matter.

First of all, the 5A/4A/4B line in
blue, the ICU line in yellow, the all others line in
purple, the 7G line in brown and the all deaths line
are all geographic lines. That is they are established
by pinpointing the place where the patient died.

THE COMMISSIONER: So that the purple
line is geographic as well on this?

MR. SCOTT: Yes. Let me just explain:
5A/4A/4B indicates that those patients died in that
ward. The ICU indicates that they died in the ICU.
The 7G indicates that they died in the 7G Neonatal
Ward. The all others is the balance of hospital
deaths that occurred in the hospital.

So that if you take the 5A blue line,
the ICU yellow line, the 7G Neonatal line and the
all others purple line, you would get the total of
all deaths in the hospital.



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THE COMMISSIONER: Then the purple line is also geographic?

MR. SCOTT: Yes.

THE COMMISSIONER: And it is only the red line that is diagnostic?

MR. SCOTT: Only the red line is diagnostic, and I think I should tell you, Mr. Commissioner, what our evidence will be as to how that is prepared.

It is based upon the diagnosis as it appears in the hospital's records under what is called the Annual Death Index Print-out, and the criteria for inclusion are all patients who died on cardiac wards, so the cardiac line includes all persons who died on cardiac wards, all congenital heart and circulatory malformations, except brain, all heart disease, all heart failure -- technically, so-called -- all heart injuries.

What it excludes are cases of cardiac arrest because, as we now know, that is the manner of all deaths.

THE COMMISSIONER: Excludes all cardiac arrests --

MR. SCOTT: Yes.

THE COMMISSIONER: -- that didn't



G7

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arise by reason of a heart --

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MR. SCOTT: With one exception. It includes cardiac arrests if the patient arrived at the hospital with that diagnosis. That is, if the patient arrived at the hospital in cardiac arrest. It excludes multiple anomalies and we will come to what is meant by that later, where the heart is not mentioned as a factor, and it excludes Down's Syndrome deaths where the heart is not mentioned as a factor.

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THE COMMISSIONER: Yes. All right.

11

EXAMINATION BY MR. SCOTT (CONTINUED)

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14

Q. Now, Dr. Rowe, we had dealt with four catagories and I want to take you to the fifth, instability of temperature.

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Is this occasionally called, in your evidence, hypothermia?

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A. Hypothermia, yes, it is.

Q. Yes.

A. It is one of the manifestations.

Q. And are there patients in the epidemic period who exhibited the symptoms of hypothermia?

A. Yes, there are.

Q. All right. Now, can you tell the Commissioner what instability of temperature is?



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A. Well, that usually means that a patient -- theoretically, it means that a patient may have a temperature that is likely to go from normal to subnormal or above normal, but in practice, the term, at least in young infants, means that they are unable to sustain a normal temperature as well as a healthy infant.

Q. All right. Now can I ask you what is the part of the body that controls or creates that -- controls body temperature or creates that condition of instability of temperature?

A. It is the brain.

Q. All right. So would it be correct to say that a child who exhibits instability of temperature is disclosing some kind of neurological or brain deficit or disorder?

A. It may be. That is often the case. The exact explanation for some babies is not that certain.

Q. Is the phenomena easily observed?

A. Yes, it is.

Q. I suppose, simply by taking the temperature?

A. Yes.



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Q. But do I understand you to say
its origin is not in every case clearly understood?

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A. No.

5

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Q. Is the brain a candidate for
the originator of this disorder?

7

A. Yes.

8

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Q. Now, can you tell us what you
know -- I should ask you more directly -- does
instability of temperature lead, in certain cases,
to a stoppage of contractions of the heart?

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A. I don't know that we know for
sure that that can occur, but in babies who have
unstable temperature there is a tendency for there to
be sudden death so I presume it has got some
relationship.

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19

20

Q. All right. Can you tell us
anything about the connection between instability of
temperature and a heart stoppage? Why shouldn't the
heart just go on pumping regularly? I'm not talking
about a normal heart. Why shouldn't the heart just
go on pumping regularly as your temperature drops?

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22

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25

A. I think the problem arises
because when your temperature drops the body makes
automatic efforts to raise it again, so it increases
the metabolic rate to bring the temperature back up



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2
3 again. Or at least that is the mechanism that is
4 homeostatic or keeps people the same temperature all
5 the time.

6 Q. Don't confuse me now. Simple
7 words.

8 A. Simple words. And --

9 Q. Did I interrupt you?

10 A. If you have -- the sort of
11 baby who has unstable temperature as a small baby and
12 the attempt to drive up the metabolic rate may be too
13 much for a baby who has got -- who is small and in
14 many instances has poor energy reserves.

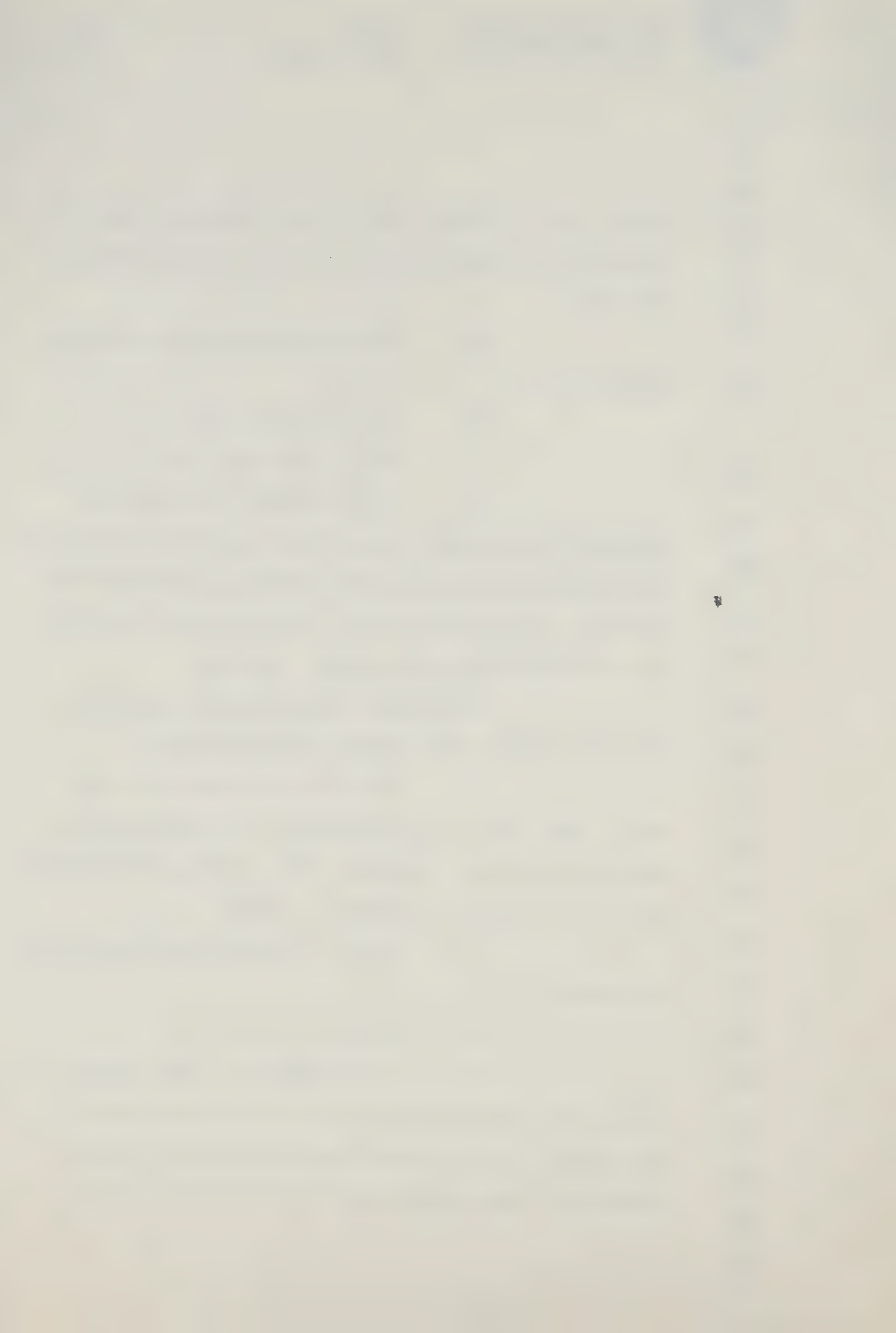
15 These are babies who are small and
16 they are not fat like chubby, bigger babies.

17 Q. You have left me with a gap
18 here. The result of hypothermia is a tendency of a
19 body to increase a metabolic rate. What, if anything,
20 does that do with the heart or lungs?

21 A. Well, it places huge demands on
22 the heart.

23 Q. Why?

24 A. The attempt to raise temperature
25 means that all energy sources are directed towards
the brain, and in the patient who has poor energy
resources, that leaves very little for the rest of





1

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the systems.

3

Q. And what is the result in a

4

clear case?

5

A. Well, that can cause cardiac

6

arrest.

7

Q. Yes. Now --

8

THE COMMISSIONER: I'm sure it can but
I still don't quite know how. How is it effecting the
heart?

10

THE WITNESS: Well, because it has

11

taken all the energy that is available to try and

12

stimulate the brain centres for temperature control,

13

and therefore robs, as it were, the other systems of

14

the body, particularly the heart.

15

Q. When this got us going,

16

in response to the increased metabolic rate to the

17

brain, I take it there is no decrease in the need of
oxygen in other parts of the body if it is to live?

18

A. No.

19

Q. What does that do to the heart?

20

A. Well, that deprives the heart

21

of the fuel, of the little fuel that is available.

22

Q. All right. So the heart is

23

deprived of oxygen by that mechanism; is that correct?

24

A. And energy.

25



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Q. Yes.

3

A. Utilization.

4

Q. That is one thing. Is the

5

demand on the heart to produce oxygen or to pump

6

increased?

7

A. Yes, it is.

8

Q. So do those two things occur --

9

A. Yes.

10

Q. In classic hypothermia?

11

A. Yes.

12

Q. And may that lead to heart

stoppage?

13

A. Yes, it may. It doesn't make

14

it always do it, but it may.

15

Q. And that can occur, I take it,

16

in babies with perfectly normal hearts?

17

A. Yes, it can.

18

Q. You have seen that?

19

A. Yes. It is a common disorder

in neonatal units.

20

MR. PERCIVAL: Mr. Commissioner, I am

21

having difficulty with the use of the word hypo or

22

hyperthermia, and I'm wondering whether or not there

23

is a distinction between the two.

24

THE COMMISSIONER: One is high and one

25



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is low.

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MR. PERCIVAL: That is right, and I am wondering whether we are talking about low temperature up to this point in time. I am sorry.

6

7

THE COMMISSIONER: Hypo is low and hyper is high.

8

9

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13

THE WITNESS: That is right.

MR. SCOTT: I take it -- I have caused this confusion, Dr. Rowe, and it won't be the last confusion I have caused -- but I take it the mechanics that we are talking about are the same whether the instability of temperature produces a high temperature or a low temperature?

14

15

16

A. Yes.

Q. The demand of the brain for oxygen

17

18

A. Yes.

Q. -- is created by either of those conditions?

19

20

A. Yes, it is, but the usual thing in small babies is hypo, but it may be hyper.

21

22

Q. And what is hypo again, just so I won't make a mistake again?

23

24

25

A. Hypo is body temperature less than normal.



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Q. You have told us this can occur
in the case of a baby with a normal heart, and I
don't want to keep repeating this question, but a
baby with an abnormal heart structure and hypotherm-
ia, what is the incidence of risk to that baby of
cardiac arrest?

8

A. I think it is additive.

9

10

Q. And I take it if you add
respiratory illness on hypoxia or sepsis, what
happens?

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A. It's the same, they all have a contribution.

Q. Yes. Now, a baby who is dying of hypothermia or instability of temperature, will that baby on the passage to brain - I'm sorry, to heart contractions stopping exhibit bradycardia?

A. Yes.

Q. Yes. Will the baby exhibit vomiting from case to case?

A. I don't know.

Q. You don't know about vomiting in that case. And when you say you don't know, just so we are clear, I take it that means you have never seen it?

A. Well, this condition is seen more often in the neonatal floor than perhaps in other parts of the hospital. So, we see it in sickly babies who are small with congenital heart disease who are not necessarily neonates. But I don't recall specific babies with vomiting, I would have to perhaps refer to the individual charts of those babies that were affected in this group we're talking about.

Q. Is the onset of arrest or deterioration a sudden one?

A. Yes, can be.



1
2 Q. Yes. Is it accompanied by
3 ventricular fibrillation?

4 A. If the baby has congenital
5 heart disease it may be.

6 Q. Yes. And you've made that
7 observation before that ventricular fibrillation is
8 usually found in babies with congenital heart
9 deformities?

10 A. Yes. The reason for the
11 difference appears to be that you need a certain
12 critical mass of muscle before fibrillation can develop.
13 There has to be a certain specific mass of muscle.
14 I don't know that there is a figure of how many grams
15 or anything like that, but that's the theory by which
16 the differences are explained between older individuals
17 who have heart stoppage and younger patients. Younger
18 patients tend to have bradycardia more often than older
19 patients. Older patients tend to have ventricular
20 fibrillation.

21 Q. Can it be accompanied by
22 arrhythmia?

23 A. Yes.

24 Q. Can it be accompanied by shallow
25 respiration?

A. Yes, indeed.



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Q. Can it be accompanied by seizures?

A. Yes.

Q. Well now, I want to take you to another characteristic to which you have referred in your evidence and that is the case of babies with low birth weights. Now, first of all, is there any connection between low birth weight per se and a stoppage of contractions of the heart?

A. Not necessarily.

Q. All right. Is it possible?

A. Well, if you have a very, very small baby who is just barely on the viable range, then that may.

Q. All right. What do you mean by low birth weight?

A. Well, a low birth weight is usually meant to - it has different definitions from different people but most I think would agree that a baby that is under 2500 grams is a low birth weight baby.

Q. All right.

A. And there are varying definitions of low birth weight. Some people talk about very low birth weight babies as being under 1500



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grams.

Q. Well, let's talk about low birth weight babies. Is there any effect of low birth weight on the brain?

A. I don't know that I can answer that question specifically but low birth weight babies are more susceptible to brain injury or brain damage.

Q. Yes.

A. So, I think that the answer would probably have to be yes.

Q. Now, what effect does that have on heart stoppage?

A. Well, I think that the baby who has a low birth weight is not necessarily, but it usually is the case that it is immature in its development; that is premature.

Q. Yes.

A. Prematurely born. But some babies have low birth weight for an appropriate age and duration of the pregnancy. So, there's a little difference between those two types of babies, as I understand from the neonatologists, they are the people who really can answer all those questions in more detail. But no doubt that in a large number of low birth weight babies the brain function has certain



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immaturity.

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Q. Yes. Those baby lack fat I

4

take it, characteristically?

5

A. They do lack fat, yes.

6

Q. Does that have any impact on

7

the need for oxygen in the baby's system?

8

A. Well, they have very little

9

in the way of energy reserves.

10

Q. And when you say energy reserves,

11

what do you mean?

12

A. I mean the fuel by which the

13

body can function is limited.

14

Q. All right. Does that have

15

any impact on the possibility of heart stoppage?

16

A. Yes, it does.

17

Q. Yes. And how does it work,

18

what's the process?

19

A. Well, the more fuel reserves

20

you have the longer it's possible to go if you are
stressed as a baby of that weight. In taking it in
its extreme form, the baby who has a good reserve of
sugar in the liver and the heart will survive

21

hypoxia for a longer period than a baby who has

22

limited reserves of those substances. I can't give

23

you all the biochemical associations of that sort,

24

25



1
2 but I think that's fairly definitely accepted.

3 Q. Well, what I'm trying to get
4 at is whether in the case of a very low birth weight
5 baby there is a connection between that birth weight
6 and heart stoppage that may exist irrespective of the
7 presence or absence of heart defect?

8 A. Yes. We're not quite sure
9 why that is but that has been an observed phenomena
10 that babies of that weight are subject to sudden and
11 unexpected death, even though the heart may be normal.

12 Q. All right. Now, I'm not asking
13 you to try and get the Nobel Prize, but can you just
14 tell us what you think as a matter of speculation the
15 connection between very low birth weight and heart
16 stoppage may be?

17 A. Well, it may be because of
18 things that occur in the respiratory centre of the
19 brain, that is, the centre that governs the breathing
20 apparatus and if that for some reason goes out of
21 kilter, then the baby could stop breathing and that
22 could end up in an arrest.

23 Q. Yes. Well now, if that
24 happens - let me put it this way. We were talking
25 about very low birth weight babies and I take it for
very low birth weight babies this can occur in the



1
2 absence of any other indicia of defect or illness?

3 A. Yes.

4 Q. But in the next category, low
5 birth weight babies, is this a factor that impacts on
6 other diseases, defects of disorders that may exist?

7 A. Oh, yes.

8 Q. So that a baby with heart
9 defect who is low birth weight, what happens to the
10 risk of cardiac arrest in that baby?

11 A. I think it's higher and I
12 think more than that if the baby's weight does not
13 increase normally in the first few months, then those
14 babies who may be of even apparently normal birth
15 weight or close to it, there is a tendency for babies
16 with congenital heart disease to have lower birth
17 weights than the average population, but given that
18 the birth weight is within the usual range and the
19 baby doesn't thrive because of the presence of the
20 heart defect, then that baby in my view begins to
21 respond more like babies of low birth weight.

22 Q. All right. So, you can in
23 substance, regardless of your birth weight at birth,
24 you may become like a low birth weight baby if you
25 fail to thrive after birth?

26 A. Yes.



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Q. Now, dealing with a case of a baby of very low birth weight with no cardiac defect who is going to die, will not the manner of dying be typically accompanied by bradycardia?

A. Yes.

THE COMMISSIONER: From what though, from the low birth weight?

MR. SCOTT: From low birth weight. Am I right about that?

A. Yes.

Q. Will it be accompanied by vomiting?

A. I'm not sure of that.

Q. All right. Is the onset of the disorder or its deterioration likely to be sudden?

A. Yes.

Q. Is it likely to be accompanied by ventricular fibrillation?

A. I don't think so.

Q. All right. Is it likely to be accompanied by arrhythmia?

A. Yes.

Q. Is it likely to be accompanied by shallow respiration?

A. Yes.



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Q. Is it likely to be accompanied by seizures?

A. It could be.

Q. Yes. And there are cases in the epidemic period of babies for whom low birth weight may be a factor leading to heart stoppage?

A. Yes.

Q. Well, now I come to the next general heading Conduction. I had difficulty with conduction as the cause of heart stoppage, so, I divided it into four categories when I discussed it with you and perhaps we can deal with them as four categories because they seem to be slightly different. The first is conduction failure type one, electrolyte imbalance. Now, can you tell me what I'm talking about.

A. Well, the ---

Q. First of all, if I can interrupt you. What are electrolytes?

A. Electrolytes are salts that are in the body normally.

Q. Yes.

A. And the chief ones that are involved are sodium, potassium and chloride.

Q. Yes.

9 Which?



Rowe, ex.
(Scott)

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A. But there are others like calcium and magnesium and other substances.

Q. Are those salts typically found in every cell in the body?

A. Yes.

Q. And what do we know about the role that those salts play in those cells?

A. Well, they are very important in cell behaviour because there is a different distribution of different substances like potassium and sodium which have different concentrations inside and outside cells. This balance of these ions and salts is critically important to the general behaviour of cells in the body.

Q. So, if I can stop you there just to see if I understand it. These salts are found in varying proportions in every cell?

A. Yes.

Q. In the body?

A. Yes.

Q. And they are in some way critical to the operation of the body's systems?

A. Yes.

Q. Now, can you tell me how they are critical?



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A. Well, you know, I am not a biochemist.

Q. In sentences of 25 words or less.

A. I'm not a biochemist and it's a long time since I did my second year of medical school but I think that the important thing is that there are relatively constant relationships of the salts and these metals and these substances like potassium and calcium and magnesium and so on inside on the membranes of cells and inside the cells and outside in fluids surrounding the cell and it is essential that they are concerned with membrane activity and there is movement of these substances across the membranes under certain conditions. If they are disturbed in any way, if the relationships and concentrations are disturbed in any way, that can vitally affect the function and the performance of any particular cell.

Q. All right, including the heart?

A. Including the muscles of the heart or the nerves of the heart.

Q. All right. Now, you've told us that that can happen if there is - how would you describe it?



12

- 1
- 2 A. A disturbance.
- 3 Q. A disturbance in their ---
- 4 A. In their concentrations.
- 5 Q. In their concentrations. Now,
- 6 is that what is sometimes called an electrolyte
- 7 imbalance?
- 8 A. Yes.
- 9 Q. Now, when there is an electro-
- 10 lyte imbalance now, you've told us that that can affect
- 11 the operation of any cells in the body?
- 12 A. Yes.
- 13 Q. And of course the heart is made
- 14 up of cells?
- 15 A. Yes.
- 16 Q. How does that imbalance, in
- 17 words of one syllable, affect the heart, or how can
- 18 it affect the heart?
- 19 A. Well, it depends on which is
- 20 the predominant imbalance. You can have an imbalance
- 21 that affects one of these substances very much more
- 22 than another; for example potassium.
- 23 Q. Yes.
- 24 A. And if you have a loss of
- 25 potassium from the body for one reason or another,
- usually from things like diarrhea and vomiting,



1
2 or you can get it from just the use of various
3 medications for the heart, like, diuretics; we've
4 talked about diuretics before here, the substances
5 that increase the amount of excretion - they are
6 drugs that increase the excretion of certain of the
7 irons and salts in the body.

8 If you get an imbalance where there
9 is too little potassium in the body, then that can
10 produce changes in the electrical stability of the
11 heart.

12 Q. What do you mean when you say
13 the electrical stability?

14 A. Well, you can have, instead of
15 having a regular rhythm you can have a ---

16 Q. And can that imbalance then
17 lead to heart stoppage?

18 A. Yes.

19 Q. And can that occur irrespective
20 of the fact that the heart is a normal heart in every
21 sense?

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A. Yes, it can. For example,
if a baby has gastroenteritis, it can very quickly
and very rapidly get into a state of potassium loss
in the body that would do just that.

Q. So, does that mean that that
imbalance of one of these electrolytes may lead to
heart stoppage?

A. Yes.

Q. And cardiac arrest?

A. Could do. Now, hopefully,
you get to babies before they do that, but it can do.

Q. Now, you have told us in a
sense how this imbalance can be caused, and I take it,
just to summarize, it can be caused by a diuretic?

A. Yes.

Q. Diarrhea?

A. Yes.

Q. How about low intake of water?

A. Or inadequate intake of
potassium in the diet. I am talking about potassium.

Q. Yes.

A. You are talking about every-
thing?

Q. I am talking about the other
two; sodium and chloride, as well.



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A. Well, you know, there are different things that can increase or decrease those.

Q. Tell us if I am covering the things.

A. Yes, you are.

Q. A low intake of water.

A. Yes.

Q. Low or inadequate feeding.

A. Yes.

Q. Vomiting.

A. Yes.

Q. Anything else that can destroy the balance.

A. Well, if you are talking about a loss of the substance?

Q. Yes, I'm talking about that.

A. You may have conditions in which the kidney is abnormal, is functioning abnormally. Kidney diseases can produce a lot of disturbance of these substances. There are a whole host of diseases that can either cause an excessive loss of these substances or can retain them in the body to an excessive level.

Q. And is this electrolyte failure, type no. 1, which we have been talking about,



I4

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characterized by vomiting?

3

A. Yes.

4

Q. Will it be marked by a

5

sudden deterioration or onset?

6

A. Yes.

7

Q. Will there be ventricular

8

fibrillation?

9

A. There may be, especially if

10

the baby has congenital heart disease.

11

Q. Will there be arrhythmia?

12

A. Yes.

13

Q. Will there be shallow respiration?

14

A. Yes. And I emphasize that

15

would be in the case of fairly severe derangement.

16

Q. Would there be seizure?

17

A. There may be.

18

Q. And are there cases in this

19

ward where there is evidence pointing to this kind of difficulty?

20

A. Yes.

21

Q. Now, let me come to conduction

22

failure type no. 2, which is the eighth cause of death.

23

Are you familiar with a conduction

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failure that results from the introduction of an

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extraneous substance?

A. Yes.

Q. Can you tell us -- can you give us a list of some of the extraneous substances which may be introduced and, thereby, cause a conduction failure type 2? How about digoxin?

A. Digoxin, I think we can say is a major contender.

Q. Anything else?

A. Potassium can do the same.

Q. Yes.

A. An excess of calcium can do the same.

Q. How about quinidine?

A. Quinidine, propanolol.

Q. How do you pronounce that?
I haven't got that right yet.

A. Propanolol, p-r-o-p-a-n-o-l-o-l.

Q. I take it that these are all natural substances, or therapeutic substances, which may get introduced to the body but which may cause electrolyte imbalance?

A. Yes. Well, I am not sure about propanolol and quinidine.

Q. I'm sorry, conduction failure.



I6

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A. Yes, conduction failure.

3

Q. And will you tell the

4

Commissioner - and, by the by, me - how it happens

5

that this may relate to, and cause, a heart stoppage?

6

A. Well, if you look at, say,
potassium--I will have to take one or two individually.

7

Q. Yes.

8

9

A. The effect of potassium, when
one gives that to an individual, will be to change the
speed of conduction of the electrical impulses in the
cardiac conduction system. So, the complexes,
instead of being, on the electrocardiogram, nice,
narrow blips and so on, will broaden out and take a
longer period to conduct, and there is sort of a rough
relationship between the levels of potassium in the
blood and the degree of electrocardiographic disturbance,
and it can progress from that to complete heart block
and so on.

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Q. What do you mean by "heart
block"?

19

20

A. Where the impulses at the top
chambers are not transmitted through to the bottom
chambers, which then begin to start their own rhythm
at a slower rate.

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Q. Now, leaving aside the reason

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I7

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why the substance is introduced to the body, can these substances, when introduced, cause a conduction failure of the type you have described?

A. Yes.

Q. And can that conduction failure lead to heart stoppage?

A. Yes.

Q. Can that occur in the case of a perfectly normal heart?

A. Yes, it can.

Q. Are you familiar with that in the literature and in the clinic?

A. Yes.

Q. Now, when that happens, when death is going to occur from conduction failure no. 2, will it be accompanied by bradycardia?

A. Yes.

Q. Will it be accompanied by vomiting?

A. It may be. I am not sure of the exact proportion in that.

Q. Will it be accompanied by, or will it be marked by a sudden onset or sudden deterioration?

A. It could be.



I8

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Q. Will it exhibit ventricular fibrillation, with the rider that you previously added?

A. Yes.

Q. Which is, I take it, that that more often occurs when there is a heart defect?

A. Yes, or when the individual is a little older than a baby.

Q. Will it be accompanied by arrhythmia?

A. Yes.

Q. Will it be accompanied by shallow respiration or seizure?

A. It may be.

Q. Or both?

A. Yes.

Q. Now, conduction failure type no. 3, Item 9, is there a conduction failure that is caused by the act of operating on a baby?

A. Yes.

Q. That is, cutting a baby open for some surgical or like purpose?

A. Yes, there is.

Q. Now, will you tell the Commissioner how that may produce a conduction failure.

A. There are several ways in which



I9

1
2 heart surgery can produce disturbances in the heart
3 rate and conduction. An obvious one, and one that
4 we sort of anticipate today will be relatively in-
5 frequent, is when the main cardiac nerve near the
6 atrial ventricular node, the portion of the conduction
7 system that passes down to supply the two pumping
8 chambers, when that is interfered with by repair of
9 a ventricular septal defect or some such arrangement
10 in the ventricles. In placing a patch to close a
11 hole, the surgeon has to put stitches around the
12 margins of the patch, and that scenario is very close
13 to where this nerve is - you can't see the nerve; it
14 looks the same as heart muscle and, so, originally, it
15 was a very difficult thing to avoid but, nowadays,
16 surgeons can - except in more complex conditions -
17 make a very good attempt to avoid that. If the
18 stitching involves the nerve or the blood supply to
19 that nerve, then it can interfere with the conduction.

18 THE COMMISSIONER: What is the
19 name of this nerve again?

20 THE WITNESS: Well, this is the main,
21 perhaps we should call it the bundle of HIS.

22 THE COMMISSIONER: I wonder if we
23 could go back to Exhibit 41A.

24 MR. SCOTT: Yes. Is that the big
25 chart?



I10

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2 THE COMMISSIONER: It is the big
3 chart, I think, yes.

4 THE WITNESS: It might be helpful
5 to have that.

6 MR. SCOTT: Is that it there?

7 THE COMMISSIONER: Yes. That is
8 something I have. I think it would be better if we could
9 find the big one.

10 MR. SCOTT: Mr. Registrar, which
11 is the exhibit, the small one or the big one.

12 THE COMMISSIONER: It is Exhibit 41A
13 that we are looking for.

14 MR. SCOTT: We can't seem to find it.
15 Perhaps Mr. Lamek can help us. It is the Pacsai chart.

16 THE COMMISSIONER: No. 41A was
17 one that -- The Pacsai chart will be just as good.

18 MR. SCOTT: Do you know where it is?

19 MR. LAMEK: No. If it is not in
20 that bundle, I don't. It is one of the very first
21 drawings of the normal heart.

22 MR. SCOTT: We have it, Mr.
23 Commissioner.

24 Q. Now, you were talking about
25 conduction failure type 3, caused by surgery --

THE COMMISSIONER: Just a moment. I



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want to make sure we know what exhibit we are talking about.

MS. CRONK: Exhibit 108, Mr. Commissioner.

THE COMMISSIONER: Yes. All right.

THE WITNESS: We are not talking about, Mr. Commissioner, either of those individuals.

THE COMMISSIONER: No. I understand that.

THE WITNESS: This is just to refresh our memories about the conduction system.

There is the sinoatrial node, up in the top portion of the right atrial or right orifice and, from there, there are impulses sent across the top chambers of the heart that then concentrate at the atrial ventricular node. From that point, there is a main bundle of HIS; then the HIS, and that is spelled H-I-S, which is a major nerve structure that looks like muscle but is a specialized muscle, and passes from that AV node until it branches at this point, at the upper part of the ventricular septum into two main branches; the right bundle and the left bundle, and these bundles are the sort of telephone wires, as it were, for impulses, electrical impulses, to travel down to the muscles of the pumping chambers.



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So that in a repair of a ventricular septal defect in this region here where the patch has to be placed, there is a specific risk that in the course of the repair the nerve may be caught with the suturing of the patch.

Now it doesn't happen very often now, but at the beginning of this surgery about a quarter of all patients have that problem. Now because surgeons know where that thing goes, they can avoid it in most instances, but there will still be a theoretic risk and there is a potential for that development.

THE COMMISSIONER: But the nerve, to distinguish it from other nerves, what would you call it?

THE WITNESS: Well, it is not really a nerve in the general appearance of a nerve anywhere else. It is really a specialized muscle. You can't tell where it is if you look at the heart.

You can only know that from examination of the hearts of children who have not survived and of hearts that were belonging to individuals who died before heart surgery became available.

THE COMMISSIONER: I take it if you cut it then all electricity ceases.



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THE WITNESS: At that point.

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THE COMMISSIONER: Yes.

4

THE WITNESS: It is like you produce

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complete heart block. It is just as though at that

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point you have completely severed the situation. It

7

produces a picture which is like congenital heart

8

block, but it is induced by the surgeon.

9

The more complicated the defect, the

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more difficult it is to be sure where that passage

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is because sometimes it is in front of the hole and

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sometimes it is behind the hole, and it takes a lot

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of specialized electrical testing, like an electrician,

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at the time of operation nowadays to be sure where

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that is.

16

In any event even interference with

17

the blood supply from the node by the stitching can

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cause abnormality of function of that conduction

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system. So that is one way.

20

If that happens there is a high

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probability that that patient would die. It is not

22

well tolerated and usually those patients have to have

23

a pacemaker inserted very quickly.

24

Any operation on the top chambers of

25

the heart can interfere with the sinus node. And

the sinus node is the real pacemaker of the heart



1
2 under ordinary circumstances. So if you interfere
3 with that you are left with pacemakers that develop
4 lower down: either at the AV node here (indicating)
5 or at various places in the ventricular myocardium
6 or muscle. And that is quite a serious condition too.

7 If it occurs, not because people put a
8 stitch necessarily through the node because they
9 usually don't go at that point, but it most often
10 seems to occur by any extensive surgery that is
11 necessary on the upper chambers can interfere with
12 the blood supply to the node. So you can have
13 disturbances of rhythm from the technique of inter-
14 ference with the blood supply or by direct damage
15 to the node.

16 MR. SCOTT: Q. Just so that I will
17 get some perspective how big is the heart of a baby?
18 That is a one month old baby?

19 A. Oh, about 28 to 30 grams.

20 Q. That doesn't mean anything to
21 me.

22 THE COMMISSIONER: About the size of
23 the baby's fist is what we have heard before.

24 MR. SCOTT: Q. About the size of a
25 plum.

THE COMMISSIONER: And a grown person's



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about the size of Mr. Lamek's fist.

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MR. SCOTT: There is not room in here for that.

5

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THE COMMISSIONER: About the size of the baby's fist, would that be fair, for the whole heart?

8

9

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THE WITNESS: Yes, that would be fair unless the heart is malformed, in which case it might be much bigger because it is thickened out and got stretched a bit.

11

12

13

MR. SCOTT: Q. Do these difficulties occur in the case only of septum repairs or any other heart surgery as well?

14

15

16

A. They can occur in any form of heart surgery because one or other - any form of intracardiac operation. Operations outside the heart will not necessarily produce that.

17

18

19

Q. How do you learn that it has occurred? Do you learn because the baby died and you do an autopsy?

20

21

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A. No, because if you get a disturbance of rhythm then it will become apparent on the clinical examination and the monitoring, especially since these children, postoperative, would go to the Intensive Care room where there



J5 1
2 would be - Intensive Care area where they would have
3 very close monitoring, electrocardiography.

4 Q. Are there any babies in the
5 epidemic period for whom this might be a consideration?

6 A. I can't recall just offhand,
7 but I could look into that.

8 Q. I am going to ask you to
9 divide the babies up with regard to these 14 cases.
10 In your spare time.

11 MR. PERCIVAL: Mr. Commissioner,
12 that question was not put on the No. 8 one as well.
13 I didn't know whether that was an oversight on
14 Mr. Scott's part.

15 MR. SCOTT: Q. The No. 8 is
16 digoxin, quinidine, propranolol, other babies for
17 whom those possibilities are a consideration in this
18 epidemic period.

19 A. Oh, yes.

20 MR. SCOTT: I presume, Mr. Percival,
21 that was in the nature of a debating point, was it?

22 MR. PERCIVAL: Oh, no, I just
23 thought it was an oversight.

24 MR. SCOTT: No.

25 MR. PERCIVAL: I am trying to be
helpful.



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J6 THE COMMISSIONER: I can do the same thing but you perhaps are not finished with this operating...

MR. SCOTT: Had you a question, sir?

THE COMMISSIONER: No. The only thing is that in every case you have asked, and certainly you must conduct your case as you want to, but whether the symptoms ---

MR. SCOTT: I haven't come to that yet.

THE COMMISSIONER: No. I just want to make sure that you did.

MR. SCOTT: I won't take as long as Mr. Lamek went about it.

MR. LAMEK: That is a very cheap shot. Not worthy of you.

MR. SOPINKA: You should have said you needed a lot of help.

MR. SCOTT: This is just the first morning, Mr. Commissioner.

Q. If death occurs from conduction failure, Type No. 3, Dr. Rowe, I take it that that conduction failure would be attributable to some defect in the heart because you wouldn't have operated



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on a normal heart.

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A. No. That is correct. It is possible for people as we have seen to have congenital heart block.

6

Q. Yes.

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A. One of the patients I believe was a patient who had congenital heart block, so you can be born with that block. There are patients who developed what we call sick sinus which means that the sinus node just doesn't function well who don't have any other disease of the heart system.

12

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Q. Let me see if I understand then. The conduction failure, the Type 3 that we are talking about, you illustrated by a reference to how the failure might be created by a surgical incident?

17

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19

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21

A. Yes.
Q. Are you telling us that it can also exist in the congenital heart?

22

23

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A. Yes, it can.
Q. And you think there may be a baby who exhibited that in this case?
A. I am not sure again exactly of the numbers here.

Q. All right. How about Bruce



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Floryn, for example?

3

A. Yes.

4

Q. Did he exhibit it?

5

A. Yes.

6

Q. All right. We will get at

7

our list later.

8

I suggested to you that this condition

9

is not going to occur in the case of a normally

10

arranged heart. Is that correct? Because it is

11

itself a defect of the very heart of the child, or

12

am I wrong about that?

13

A. No, I think - I am not quite

14

sure of your question.

15

THE COMMISSIONER: Remember we

16

started off this was an operation.

17

MR. SCOTT: Yes.

18

THE COMMISSIONER: So it is in the

19

operation, and I thought it was ---

20

MR. SCOTT: No, Dr. Rowe has gone

21

on to say that an operation is one way it may be
caused. It may exist congenitally. That is there
may be - it is not natural but there may be at birth
a block.

22

THE WITNESS: Yes, or it may be

23

acquired.

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MR. SCOTT: Q. How can it be
acquired?

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A. It probably has its origins
farther back than that but you may for the first time
see somebody who has had heart block that develops
spontaneously at, say, four or five days of age.

8

9

Q. So heart block is the
description of this phenomenon, is it?

10

11

A. Yes.
Q. Do I have it right then that
heart block can onset unexpectedly?

12

13

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A. Yes.
Q. Be congenital?
A. Yes.
Q. Or be caused by surgery?
A. That is correct.
Q. And if that happens what is
the result in relation to the contraction of the
heart?

19

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A. Well, the heart can contract
fairly well in a patient who has heart block, complete
heart block that occurs spontaneously without any
underlying disease. Then what happens is that the
contraction of the heart is a bit like a mile runner
or something, some other distance in the new system.



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The heart is slow and the heart enlarges to cope with
a need to deliver the same amount of blood per
minute, but at a slower beat.

5

Q. Yes.

6

A. And it contracts fairly

7

effectively.

8

The problem is that in heart block

9

that is either surgically created or that is - and

10

in many patients at least with congenital heart block

11

the rate is so slow that there is an opportunity for

12

you get ectopic rhythms or irregularity as well as

13

slowness.

14

Q. And what does that slowness

15

plus the irregularity created by other pacemakers

16

lead to?

17

A. Well, you can die suddenly

18

with that.

19

Q. Well, when you die suddenly --

20

A. Ventricular fibrillation.

21

Q. Are we talking then about a
heart stoppage?

22

A. Yes, indeed.

23

Q. And would that be accompanied

24

by bradycardia?

25



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3 A. Yes.
4 Q. I think you have perhaps
5 answered it.
6 A. Yes.
7 Q. Would it be accompanied by
8 vomiting?
9 A. It might.
10 Q. Would it onset suddenly or
11 would the patient deteriorate suddenly or unexpectedly?
12 A. Could do.
13 Q. Would it be and perhaps you
14 have answered this, accompanied by fibrillation?
15 A. Yes. Not always but it may be.
16 Q. Would it be accompanied by
17 arrhythmia.
18 A. Yes.
19 Q. Would it be accompanied by
20 shallow respiration?
21 A. It may be.
22 Q. Would it be accompanied by
23 seizure?
24 A. Could be, too.
25 Q. No. 10, conduction failure
Type 4, viral infection of the heart. Are you familiar
with that expression?



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A. Yes.

3

Q. What does it describe?

4

A. It describes an inflammation

5

of heart muscle with a virus.

6

Q. Well, just explain that to me.

7

A. Well, virus of the sort that

8

everybody knows, Cocksackie viruses and a number of
others can cause ---

9

Q. One of those wiggly things

10

you see on a microscope slide?

11

A. Not too wiggly; you can't

12

see them very well except with an electron microscope.

13

But this is a virus, a small particle, a small

14

infective particle that could start off as an

15

apparently benign infection like a cold or a flu

16

type illness and affects the heart muscle and

17

damages the heart muscle to the effect that it

18

causes death of parts of the heart muscle and death

19

Q. Just if I can stop you there.

20

Is that just as a muscle may get a viral infection or

21

an illness, an arm muscle or a leg muscle, are you

22

saying that the heart muscle can get that illness as

23

well?

24

A. Yes.

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Q. And that is in the case of a perfectly normal heart?

A. Yes.

Q. Yes. Now what happens if the heart gets an illness in that fashion?

A. Well, if it is a very wide-spread infection of heart muscle, that is an extremely dangerous condition because the heart is likely to either fail or for the conduction system to go completely out of whack very abruptly.

Q. So what you say is that if it is serious it may lead the heart to stop contracting?

A. Or to fibrillate.

Q. Let's get it stage by stage. First of all, will it stop the heart from contracting?

A. It would be more likely to fibrillate.

Q. All right. Are you aware without going back over them whether there were any cases in the epidemic period which exhibited the indicia of conduction failure Type No. 4?

A. That is with infection?

Q. Yes.



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A. No, I can't recall. I

don't think there were.

Q. All right.

Let me ask you this about -- well,
perhaps I will just go over my list first.

If death occurs as a result of an
infection in the heart, conduction failure type No. 4,
will it be accompanied by bradycardia?

A. It may be, yes.

Q. Vomiting?

A. Yes.

Q. Sudden deterioration or
onset?

A. Yes.

Q. Ventricular fibrillation,
I think you have told us about.

A. Yes.

Q. Arrhythmia?

A. Yes.

Q. Shallow respiration?

A. Yes. It can have seriousness
because, very frequently, if you have that sort of
an infection, it also affects the brain.

Q. Now, so far, Doctor, in addi-
tion to our first case of heart failure, we have dealt



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with some ten other cases of heart stoppage, nine of which may operate in the case of a perfectly normal heart. Have I got that right? You will take the number 10 from me?

A. Yes, can occur.

Q. All these, except heart failure, may occur in the case of a perfectly normal heart?

A. Yes, I'm losing track of the numbers.

Q. Well, we're down to ten with conduction failure. Can conduction failures be identified by post mortem?

A. It is extremely difficult to identify them; most cases not. In order to identify an abnormality of the conduction system, say, of the congenital type - because that is the one where most of the work has been done - an incredibly large number of sections of the heart system, the heart conduction system, has to be made. It is so big a job that most pathologists wouldn't even begin to take it on, and there are only a very few people who have had the courage to undertake this, because it must takes months to evaluate.

Q. Well now, let me come to



K3

Item No. 11, acidosis.

Is acidosis, independently of heart defect, a cause of heart stoppage and cardiac arrest?

A. Yes.

Q. Yes. Is it well-known in the clinic?

A. Yes, it's well known, particularly in the neonatal field.

Q. Yes. And would you describe to the Commissioner what is meant by "acidosis".

A. Acidosis is really a situation where the body fluids are too acid. The body fluids are normally in a neutral state in terms of acid or alkaline, and one can, through disturbances of various sorts, become either alkaline or asidotic.

Q. Is there a balance that is required?

A. Yes, there is.

Q. And is that balance as between acidine and alkaline required for every cell in the body?

A. Yes, it is.

Q. And when the balance is disturbed in favour of acid, is that called asidosis when it appears in that cell?



K4

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A. Yes.

3

Q. And can it appear in groups

4

of cells and in organs?

5

A. And in the blood.

6

Q. And in the blood?

7

A. Yes.

8

Q. Now, what happens when there

9

is acidosis of the blood or the liver or the kidney -
let's not talk about the heart just yet? May that

10

have any impact on heart stoppage in a baby?

11

A. When you have acidosis, it

12

implies that all the organs in the body will be in
the same situation. It is unlikely that one organ

13

will be less acid than another.

14

Q. I see.

15

A. That's a sort of broad

16

generalization. So that it has profound effects on

17

every cell and, of course, it depends on the key

18

organs of the body. If you have it on your toe, it

19

may not be as bad as having it in the brain, you know,

20

that sort of impact, but it depends on how vital the

21

organ is. If the organ is vital, the effect is the

22

same on cells; that is, it interferes with the functions
of the transport of substances in and out of the cell,

23

on the membrane on the internal working of the energy

24

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K5

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2 arrangement inside the cell and just the sort of things
3 we've talked about with the boxing.⁷

4 Q. And is acidosis a candidate
5 to induce the stoppage of heart contractions?

6 A. It can, indeed.

7 Q. How does it work? How does
8 that happen?

9 A. Well, again, it affects the
10 cellular action - that is the important thing. If
11 the cell becomes so acid that it cannot perform its
12 normal function, then the cell stops functioning.

13 Q. And are there cases that
14 point to that in the epidemic period?

15 A. Yes.

16 Q. Can that happen and lead to
17 heart stoppage in the case of a perfectly normal
18 heart?

19 A. It can. It would have to
20 be very extreme, but it can.

21 Q. Is there, again, the build-up
22 if you have acidosis plus a heart defect that the
23 risk to the patient is greater than with the heart
24 defect alone?

25 A. Yes.

Q. Now, when a patient has



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acidosis, you have told us it can lead to the stoppage of contractions. Is that accompanied by bradycardia?

A. Yes.

Q. I'm getting this out of order. First of all, can this theoretically occur where there is no heart defect?

A. It can, yes.

Q. But are you telling me it is more likely to occur where there is a heart defect?

A. No. It can occur in small babies who are ill or for any different number of reasons, but it is particularly likely to occur in babies who have lung problems or who have some metabolic upset and the patients with heart disease, where there is heart failure, there is usually a problem with the distribution of blood around the body and, therefore, acidosis is a fairly common thing. In some patients with congenital heart disease, it is even worse than that. If you have a ductal-dependent baby - that is, one where the presence of the open ductus arteriosus is critical for survival - if the conductus shuts off, one of the first things that happens is that you get metabolic acidosis because the blood is not being profused around the heart, and the moment that starts, it is an inexorable course and the



K7

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patient will become acidotic metabolically and that
just shuts down everything.

4

Q. And that leads to death?

5

A. Yes.

6

Q. And that leads to death,
again by the stoppage of contractions?

7

A. Yes.

8

Q. And cardiac arrest?

9

A. Yes.

10

Q. And apart from the ductus
that you described, there may be nothing else wrong
with the heart?

12

13

A. Oh, no, there usually is.
I'm talking about the ductal-dependent heart lesions.

14

Q. I see.

15

Now, when that occurs, is it likely
to be accompanied by bradycardia?

17

A. Yes.

18

Q. Is it likely to be accompanied
by vomiting?

19

20

A. It may be.

21

Q. Is it likely to onset
suddenly or the patient to deteriorate suddenly?

22

A. Yes.

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Q. Is it likely to be accompanied

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by ventricular fibrillation?

A. If there is congenital heart disease, yes.

Q. Yes. Is it likely to be accompanied by arrhythmia?

A. Yes.

Q. Is it likely to show shallow respiration?

A. Yes.

Q. Is it likely to produce seizure?

A. Yes.

MR. SCOTT: Would this be a convenient time, Mr. Commissioner?

THE COMMISSIONER: Yes, until 2:30.
--- luncheon recess.



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--- on resuming.

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THE COMMISSIONER: Yes, Mr. Scott.

4

MR. SCOTT: Just one matter.

5

When Mr. Lamek was examining Dr.

6

Rowe on July 28th, Volume 18, beginning about page
3077, he drew to his attention a note --

7

MR. LAMEK: I'm sorry, what page?

8

MR. SCOTT: Well, it was dealt

9

with in various places but I think it is dealt with
at page 3077.

10

11

MR. LAMEK: Thank you.

12

MR. SCOTT: He drew to his attention

13

a note, I think in the Woodcock record, which read:
"could possibly be some sort of drug overdose,
accidental or otherwise".

14

15

You will remember that that was the

16

case of the Baby Laura Woodcock, who was I think

17

jaundiced and a doctor whose signature couldn't be

18

identified had made that note in the record or in the

19

chart, and Mr. Lamek drew from that that there was,

20

in June at least, the author of that note who was

21

prepared to contemplate the possibility of intentional

22

overdose of some drug and as a possible explanation,
presumably, of the death of the child.

23

Dr. Rowe didn't read the note that

24

25



1
2 way but Mr. Lamek apparently in the ensuing questions
3 did.

4 Now, I should tell the Commissioner
5 that we have made an effort to find out who made that
6 note, notwithstanding that his signature gave us
7 almost no clues, and we have ascertained that it is
8 a Dr. Webber, who has now returned from his vacation.

9 So that a false impression about
10 the impact of that note won't remain with you, Mr.
11 Commissioner, or with the public or press, I would
12 ask Mr. Lamek to - and we'll be glad to assist -
13 interview that doctor as soon as possible and to call
14 him out of order if necessary because the imputation
15 that his question made out is a serious one, and I
16 think it can be put to rest if Dr. Webber is inter-
17 viewed.

18 MR. LAMEK: Mr. Commissioner, Mr.
19 Scott has already told me about Dr. Webber's avail-
20 ability for interview and, of course, I will inter-
21 view him. So far as the imputation is concerned, I
22 refer my friend to the passage on page 3078 where
23 Dr. Rowe said to me:

24 "I don't believe he's referring to
25 the mode of death, he's referring to
the liver."



1
2 And my question was:

3 "Q. Whatever he's referring to,
4 Doctor, he contemplates as a possible
5 explanation, does he not, intentional
6 drug overdose?"

7 In my respectful submission, the
8 imputation my friend suggests was plainly not left,
9 but it should be cleared up anyway, and I will inter-
view Dr. Webber.

10 MR. SCOTT: That makes the point
11 nicely, yes.

12 THE COMMISSIONER: All right. And
13 you will make him available and then this next
14 problem we don't have to face until after that inter-
view.

15 MR. LAMEK: That's correct.

16 MR. SCOTT: Well, one has diffi-
17 culties because there has been all this talk about
18 a mystery doctor and I just want to make sure there
19 is no mystery.

20 THE COMMISSIONER: He is no longer
21 mysterious! All right. That's fine.

22 MR. SCOTT: Q. Well now, Dr.
23 Rowe - if I may continue, Mr. Commissioner - we had
24 dealt with acidosis before lunch. I want to take you
25



1
2 to a word that you have used and which might form
3 the heading for my next series of questions - Apnea,
4 A-p-n-e-a.

5 Can you tell the Commissioner what
6 apnea is.

7 A. Apnea is the absence of
8 breathing.

9 Q. Yes. And what is the
10 origin of the absence of breathing in apnea?

11 A. It is probably a central
12 origin; that is, that there is a signal from the
13 brain that stops the respiratory drive on the
14 centre that is responsible for the rhythmic intervals
15 between breaths.

16 Q. Would that be characterized
17 as a neurological defect or abnormality?

18 A. Yes.

19 Q. And do I understand from
20 your answer then that the neurological defect or
21 abnormality signals the breathing to stop?

22 A. Yes. It may be a primary
23 or secondary brain --

24 MR. PERCIVAL: Mr. Commissioner,
25 may I compliment the technician who obviously did
wonderful miracles over the lunch hour because we can



1
2 all hear very well now.

3 THE COMMISSIONER: Well, that's
4 good. I'm glad that we're pleased with somebody or
5 something.

6 MR. SCOTT: If we can get Mr.
7 Percival listening!

8 MR. PERCIVAL: Mr. Commissioner, I
9 just woke up!

10 THE COMMISSIONER: Well, I will
11 pass on your compliments.

12 MR. SCOTT: That's why Dr. Rowe
13 and I will be booming at each other for the rest of
14 the afternoon!

15 THE COMMISSIONER: I don't discover
16 things as fast as Mr. Percival and I probably would
17 have done just that.

18 MR. SOPINKA: I thought it was just
19 that Mr. Scott had lunch and he had more energy!

20 MR. SCOTT: Well now, back to the
21 case.

22 Q. Is apnea capable of existing
23 so as to stop breathing altogether apart from the
24 condition of the heart or its muscle?

25 A. Yes.

Q. And is it, in the clinic and



1

2

the literature, a well-known phenomenon?

3

4

A. Yes, it is, especially in
the context of sick infants.

5

6

Q. Yes. Does this have any
relation to the blue baby syndrome?

7

8

A. Not necessarily, no.

9

10

Q. I see.
Are there any cases of the 36
through which Mr. Lamek took you that suggest the
presence of Apnea as a cause or potential cause?

11

12

A. I seem to recall that there
are.

13

14

Q. Yes. Well, would you make
a note to put this on your list.

15

16

THE COMMISSIONER: But apnea, is
it a cause in itself? It is not a symptom of something
else?

17

18

THE WITNESS: It may be a symptom
secondarily of something else but its origin is almost
always attributable to the brain.

19

20

21

22

MR. SCOTT: Q. Well, what I am
getting at though is that a baby's heart may stop
contracting if it exhibits the neurological defect or
abnormality known as Apnea?

23

24

25

A. Yes.



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Q. Which neurological defect
or abnormality can lead to a stoppage of breath?

4

A. Yes.

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Q. And it has nothing to do with
the formation or any defects in the heart?

A. It may have nothing to do with
it at all.

Q. This defect may lead to a
perfectly normal heart arresting?

A. Yes.

Q. And when that occurs is it
accompanied by bradycardia?

A. Yes.

Q. Is it accompanied by vomiting?

A. I don't think so, particularly.

Q. May its onset be sudden, or
the deterioration of the patient be sudden?

A. Yes.

Q. May it be accompanied by
ventricular fibrillation?

A. That would be unusual, I would
think.

Q. Arrhythmia?

A. Arrhythmia, yes.

Q. Shallow respirations?

A. Well, there is no respiration.

Q. No respiration at all?

A. No.



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Q. And seizure?

A. Yes.

Q. One matter, while I have you here, one of Mr. Lamek's list is, of course, vomiting. I wonder if you could tell us something about how vomiting occurs. Most of us, I think most lay persons associate vomiting with having consumed something that upsets your stomach, or perhaps having the flu or some disorder of that type. Is that how vomiting in babies occurs exclusively, or is it -- or are its origins different?

A. There are many origins to it but it can occur because of a local upset in the stomach. It can occur just if you happen to have an infection. It can occur if you have something wrong with the brain.

Q. Can it occur because you have a heart stoppage, just as a seizure might occur because of a heart stoppage?

A. You may have a terminal episode where stomach contents are excreted anyway, vomiting, I suppose is a reasonable word for it, but it may not be a projectile thing, it may just be regurgitating.

Q. Item 13, Dr. Rowe, anemia, you have used that word from time to time. Will you



BB3

1
2 describe to the Commission what you mean by anemia.

3 A. Well, anemia is a shortage of
4 hemoglobin in the blood, or the capacity of the blood
5 to carry oxygen.

6 Q. Is it a disease?

7 A. Yes. It is a disease, because
8 normally you have a certain amount and anemia is the
9 reduction below that.

10 Q. And does anemia in small babies
11 cause a stoppage, or may it cause a stoppage of the
12 heart or cardiac arrest?

13 A. It may if it is very severe.

14 Q. Would you tell the Commission
15 the mechanical process by which a disease of the
16 blood, the oxygen carrying capacity of the blood will
17 lead to a stoppage, or may lead to a stoppage?

18 A. Well, that is because oxygen is
19 essential for the activity of most metabolic things
20 going on in cells. The actual work of the cells
21 depends upon oxygen. If there is an oxygen shortage
22 and especially very severe, as can occur with some
23 types of anemia in very small and particularly new-
24 born babies, then there can be interference with the--
25 solely in the function, and that will affect, as before,
any organ, but the organs, the vital organs like the
heart



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and the brain are most likely to be affected.

Q. And may anemia, as a cause or contributing to a cause, so act, even if there is a normal heart?

A. Yes, if you have severe anemia then the heart can be damaged by the lack of oxygen, and so, the actual disintegration of cells.

Q. Can it lead to a stoppage of the heart, or cardiac arrest?

A. Yes it can, if severe.

Q. If it did, would that be accompanied by bradycardia?

A. Yes.

Q. Would it be accompanied by vomiting?

A. I don't know.

THE COMMISSIONER: I am sorry.

THE WITNESS: I don't know.

Q. Would its onset be sudden, or the deterioration sudden?

A. It could be.

Q. Would there be ventricular fibrillation?

A. Unlikely.

Q. Would there be arrhythmia?



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A. Yes, there would be arrhythmia. There might be ventricular fibrillation because there can be actual death of muscle cells in the heart and that might be a source of ectopic activity of that sort, but generally speaking, ventricular fibrillation is unusual in small babies.

Q. Would there be shallow respirations?

A. Yes.

Q. Would there be seizure?

A. There may be.

Q. Now, No. 14 in DiGeorge Syndrome, that was an expression used, or a name that you use in examination in chief.

I wonder if you could tell the Commission what the DiGeorge Syndrome is.

A. The DiGeorge Syndrome is the collection of abnormalities that is occasionally found in young babies in which there is an absence of the thymus gland.

THE COMMISSIONER: I'm sorry, absence of the sinus did you say?

THE WITNESS: Thymus, T-H-Y-M-U-S gland, and a shortage of the parathyroid gland, that is absence or hyperplasia of the thyroid gland. This



BB6

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2
3 gland is concerned with calcium metabolism, and the
4 thymus, of course, is important in the immune system
5 of the body.

6 ? In addition, the babies very frequently
7 have severe conofrontal abnormalities of the heart,
8 which means they have severe defects like truncus-
9 arteriosis, interrupted aortic arch, tetralogy if
10 you like. It is a collection of conditions in which
11 there is a threat to the patient from the heart
12 disease itself. There is a threat because of the
13 influence on calcium metabolism because they often
14 have convulsions in the new-born period because of
15 lowering of the blood calcium. In addition, they are
16 regarded as being particularly susceptible to
17 infection because of the immune system being
18 incompletely present. There is a tendency for those
19 babies to die suddenly and unexpectedly, but they
20 have enough problems there to account for a high
21 proportion of deaths.

22 Q. And what is the mechanical
23 route by which the heart stops when the DiGeorge
24 Syndrome is present?

25 A. There may be a relationship to
the calcium level in the blood because that disturbs
the electrolyte situation so that then the calcium



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gets low and the patient develops heart block.

3

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Q. Is it possible that the
DiGeorge Syndrome is perhaps more parallel to a
conduction defect?

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A. I don't know that we can say
that because they very frequently have a serious
heart disease. I think, in most cases, the effect of
the non-conduction would be additive.

10

11

Q. Theoretically, is the DiGeorge
Syndrome capable of existing and causing death when
there is a normal heart?

12

13

A. Yes.

14

Q. Is it accompanied, is death
from this syndrome accompanied by bradycardia?

15

16

A. Yes.

Q. Vomitting?

17

A. It might be in some situations,
especially if they have low calcium.

18

19

Q. Sudden deterioration in the
patient, or sudden onset?

20

21

A. Yes, it has been recognized in
the literature that they may die quite unexpectedly.

22

Q. Ventricular fibrillation?

23

A. I doubt it.

24

Q. Arrhythmia?

25



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A. Yes.

3

Q. Shallow respirations?

4

A. Yes.

5

Q. Seizure?

6

A. Yes.

7

Q. And are there cases among the

8

36 that you and Mr. Lamek reviewed, which point to the DiGeorge Syndrome as a cause?

9

A. Yes, there are two cases of

10

partial DiGeorge Syndrome, I believe, and that is variable with the degree of severity.

11

12

Q. I think you have listed for me

13

14 cases of death, causes of heart stoppage. The

14

first was heart seizure, which connotes a defective

15

heart. Do I have it right that the other 13 can

16

cause heart stoppage, cardiac arrest, even in the

17

case of a normal heart?

18

A. Yes.

19

Q. Do I have it also that when a

20

number of these -- when one of these 13 diseases or

21

disorders exists in connection with a congenital

heart abnormality, that the risk of death is greater?

22

A. Yes, I believe that is the

23

case.

24

Q. And that the risk of death may

25



1
2
3 escalate as other of these causes or diseases are
4 found present?

5 A. Yes, unless they are treated
6 very rapidly and promptly.

7 Q. Now, I want to just run through
8 them again to see the extent to which their existence
9 can be determined after death by postmortem. So
10 perhaps you can just follow with me and I will read
11 them out and you will tell me if postmortem is
12 likely to show the existence of the disorder.
13 Because I always like to begin with the easiest,
14 heart failure, would the post mortem show the defective
15 heart?

16 A. It usually will.

17 Q. How about hypoxia?

18 A. You mean signs of hypoxia on
19 postmortem or just on the heart?

20 Q. When you have a death, is
21 there anything that you can look at at postmortem
22 that will help you in determining whether hypoxia
23 played a role in that death?

24 A. Yes, there are certain
25 findings at postmortem that are said to be
characteristic of hypoxia.

Q. How about sepsis?



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3 A. You would expect to be able
4 to find sepsis unless it was extremely early.

5 THE COMMISSIONER: I'm sorry.

6 THE WITNESS: Unless the death
7 occurred very, very rapidly after the onset.

8 Q. What would you expect to find?

9 A. You might not find anything
10 there then, but ordinarily you would find evidence of
11 infection somewhere in the body, inflammatory cells,
12 destruction of heart tissue and so on.

13 Q. How about respiratory illness,
14 pneumonia, atelectasis, congestion, the airway
15 obstruction, would you likely at postmortem find
16 anything that tends at points to those?

17 A. You would find evidence of
18 pneumonia, fluid, congestion in the lung, pulmonary
19 edema and those things are fairly standard. It would
20 depend how complete the autopsy was as to whether you
21 would find evidence as to whether there was obstruction
22 and you might have difficulty if they forgot to look
23 at the back or the oropharynx to see where the edemae
24 are and that sort of thing.

25 Q. Yes. No. 5 was instability of
temperature or hypo- or hyperthermia. Would you find
evidence of that at postmortem?



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A. I would think not.

3

Q. The next was low birth weight.

4

I take it, if you weighed the baby that would be what
5 you would find?

5

6

A. You would find if the death
7 occurred shortly after birth, you would have evidence
8 of a low weight, that would give that clue.

8

9

Q. Would there be any evidence
10 in the postmortem itself, apart from the weight of
the child and its age?

10

11

A. I'm not aware of any, there
12 might be others but I am not aware of them.

12

13

Q. And I think you have already
14 told us that the four kinds of conduction failure I
15 discussed with you would not be revealed on a
16 postmortem.

16

17

A. Very unlikely to be.

17

18

Q. Without the kind of testing
19 you have described this morning?

19

20

A. And even then you wouldn't
20 find it in some of the forms.

21

22

Q. No. 11 was acidosis, is a
22 postmortem going to reveal whether acidosis played a
23 part in the death?

23

24

A. I don't believe so.

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Q. What about apnea?

A. No. That's not quite true.

Apnea, you would not be able to identify that apnea
had occurred in an individual case, unless it was
something like Sudden Infant Death Syndrome that has
a set of pathological findings, which, I believe,
are considered by many people to be characteristic.

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Q. Yes, but I take it for apnea
plain and simple apart from SIDS ---

A. Yes.

Q. --- would the post mortem
reveal anything that would point to apnea as a cause?

A. No. You might be able to see
some toxic signs.

Q. Well, what about anemia?

A. Anemia, you might be able to -
you might be able to detect that from the appearance
histologically of a heart but I am not sure what
degree of certainly one would have. I think you
would have to get a pathologist to answer that one.

Q. All right. Now what about
the DiGeorge Syndrome?

A. Yes. That could be identified.

Q. So I haven't toted them up,
but I take it a number of these causes which may cause
or contribute to this stopping of contractions in the
heart are not causes that can be ascertained post
mortem?

A. Yes, some of them, yes.

Q. Now, when you are looking for
the pre-mortem you have a number of aids in that
detection process, don't you?



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A. Yes.

Q. What are those aids just in brief?

A. Well, the low birth weight baby is obviously. Conduction failure would be the electrocardiogram principally.

Q. Yes.

A. And the instability of temperature would be the observation that the nurses would provide on a regular basis.

Q. Yes.

A. And acidosis and sepsis could be confirmed by measurements of blood Ph, and culturing blood and other fluids of the body to see if there is any infection growing. And anemia can be determined by blood count.

Q. And a catheter can determine the ---

A. And the hypoxia would be determined by the measurement of oxygen in the blood.

Q. Yes.

A. Oxygen measurement, and heart failure can be assessed by suitable assessment.

Q. Apart from those aids to detection in the clinic does the observation of the



1
2 cardiologist, the internist, the fellow, play a
3 part in assessing the extent to which these conditions
4 may exist?

5 A. Yes, I think it does.

6 Q. Can you explain how that is
7 done? It is a matter that is foreign to lawyers,
8 perhaps, but how do you do it?

9 A. Well, you do it part by
10 obtaining historical information about the patient.

11 Q. Yes.

12 A. That is you learn something
13 from the family as to what has been going on in the
14 immediately preceding time, or if the patient is
15 transferred from another hospital, from that hospital's
16 observation.

17 Then you make a physical examination
18 which tells you rather substantially more than just
19 that there is severe heart disease because it will
20 allow you to assess the severity, and there are a
21 number of ways that you can assess the severity of
22 the heart failure in addition to the severity to the
23 underlying heart disease.

24 Q. We will be coming to them
25 later but ---

A. Yes.



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Q. --- is the rest a matter of judgment on the part of the clinician?

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A. Judgment and the correct utilization of the other investigative means at his disposal.

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Q. Well now there is just one point I want to make with respect to those 14 modes by which contractions of the heart may stop.

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Have you any observation in the case of young babies as to whether their onset may be sudden and unpredictable? Let me show you the background and you will perhaps remember.

13

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16

We have had a lot of evidence that the nurse's note indicates that Baby X is stable or nothing much has been happening for a day and then there is a cardiac arrest suddenly out of the blue in a stable course.

17

18

With respect to these 14 causes does that strike you as odd or unusual?

19

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A. I think that babies can appear to be stable when they are not really stable and they can certainly deteriorate, some of them, there is absolutely no question about that, but it depends... Not always, but it depends very frequently on observation that is rather specialized in order



1
2 to determine whether those babies are as stable as
3 you might think prior to the deterioration.

4 That is because not everything that
5 is under external observation by, say, a nurse or
6 parent, would necessarily be sufficient guide to
7 indicate a deteriorating, an infant who is deteriorat-
8 ing who could appear stable on the outside.

9 Q. Yes.

10 A. And that is a difficult problem
11 because it obviously - it would demand for the thing
12 to be satisfied in terms of detection of that decay
13 would be somebody who would be knowledgeable like the
14 physician, pediatrician or cardiologist in this
15 particular area, being able to make observations that
16 are different from those that I have described by
17 others.

18 Q. Now, Doctor, I want to turn
19 to - we dealt now with 14 causes of heart stoppage,
20 and I want to now turn to a list I have made apart
21 from those 14, of other complicating factors, and I
22 will tell you and the Commissioner what I would like
23 to deal with and then I would like to ask you some
24 questions under each of these heads.

25 The first is I would like to deal
with one's capacity to measure the severity of cardiac



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malfunction.

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Then I would like to deal with extra-cardiac malformations.

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Then I would like to deal with the size and age of babies at time of birth as a complicating factor. The last, with the failure to grow and thrive in weight and height as a complicating factor.

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Now first of all, are you familiar with a study called The Report of The New England Regional Infant Cardiac Program which I think was made in February of 1980?

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A. Yes, I am.

Q. Yes. And we have copies of that. Perhaps I can show you the original and work from a copy.

THE COMMISSIONER: I take it you are familiar with it?

THE WITNESS: Yes, I am.

THE COMMISSIONER: Do you want the whole thing in, Mr. Scott? You want the whole thing in?

MR. SCOTT: Yes. This is really only part of it, is it not, Dr. Rowe?

THE WITNESS: What I have is the



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whole thing. ,

MR. SCOTT: Q. Yes. I am sorry.

It is published in a periodical known as "Pediatrics".
Is that correct?

A. I hope the copyright has all
been satisfied.

Q. Well, you leave that to us.
We copied it; not you. Just a couple of questions.

THE COMMISSIONER: Exhibit 126.

MR. SCOTT: Thank you.

---EXHIBIT NO. 126: Publication entitled "Pediatrics",
dated February, 1980.

MR. SCOTT: Q. As I understand it,
and I have read this document with a very low level
of comprehension which will be apparent, but I take
it its program, just to lead you a bit for the moment,
is a program that is run by all the hospitals in
I think ~~six~~ New England States?

A. Yes, it is.

Q. Yes. And that between 1968
and 1974 they reviewed with appropriate follow-up,
which is why they only went to 1974, some 2,251 babies
with cardiac problems.

A. Yes.

Q. Now can you describe - I'm



1
2 going to take you to some of the particular parts of
3 it, but can you just describe to the Commissioner
4 what you regard as the key findings of this study?

5 A. Well, this was a move in
6 New England to try and regionalize the care of
7 babies who were critically ill with heart disease
8 in the first year of life; therefore it concerns only
9 babies who come under that definition.

10 Q. The definition is New England
11 babies?

12 A. Yes.

13 Q. Admitted to one of the
14 treating hospitals?

15 A. Yes.

16 Q. And in the first year of life?

17 A. In the first year of life but
18 with certain qualifications about the severity of
19 their heart disease.

20 Q. Fine.

21 A. These were - I believe that
22 they - I think they had to undergo cardiac cathe-
23 terization and endocardiography, be operated upon
24 or have died to be included in the total.

25 In other words they would not admit
a baby who was quite well with a minor defect. So



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these were - they call them critically severe meaning that they were severe enough that they either died, had to have an operation or had undergone catheterization or death.

6

Q. These were the babies that

7

they reviewed?

8

A. Yes.

9

Q. And does the study reveal

10

anything about the survival rates or capacities of those babies?

11

A. Yes, it does.

12

Q. Can you tell the Commissioner

13

what it discloses?

14

A. Yes. It summarizes the fact

15

that the mortality is between 35 and 40% in all these infants.

16

Q. Now just let me see if I

17

understand that. Does that mean that of the babies

18

in this category who were sufficiently ill to undergo

19

catheterization or surgery, 35 to 40% died or 35 to

20

40% died in the first year?

21

A. Died in the first year I

22

believe.

23

Q. All right. So the study

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doesn't tell us to what extent babies in this

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category die in succeeding years?

A. Not directly I think. There may be some tables on that but I think their conclusion is concerning the first year.

Q. Now did it tell us anything about the various determinants for survival?

A. Yes. This was one of the important parts of the examination. They looked at factors that might be important in the survival and came to conclusions as a result of that analysis.

Q. Can you summarize the conclusions for us?

A. Well, as would be no surprise to anybody, the extent of the anatomic abnormality was of considerable importance.

Q. Well, if I can stop you there, did they make an effort to characterize the severity of the anatomic disorder?

A. Yes. They did do some grading and did analysis of the severity in that way I believe.

Q. And did they provide any figures as to the extent to which a severe anatomic disorder led to death apart from the 35 to 40% in the first year?

A. Yes, they did that for certain



CC11

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malformations. For example, transposition of the
great arteries.

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Q. And what did they find out?

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Is there a chart that you can point us to?

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A. I think there is a chart on
Table 27 on page 401.

Q. Yes.

A. That just gives the dif-
ferent malformations of the heart and the first
month's mortality and the first year's mortality.

It is not quite what you are asking
for but that is the first table I can see.

Q. Well, let's see if I can
read that.

What it shows is -- it lists the
various kinds of heart deformations down the left-hand
side of the page.

A. Yes.

Q. And then it lists the number
of infants in the study who were shown as disclosing
that kind of disorder.

A. Yes.

Q. And then the third column,
do I read that as the number from that category who
died in the first thirty days?

A. Yes. This is in relation to
surgery.

Q. Yes.

A. So, it is not quite as --



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2 They have tables on the left-hand column for
3 mortalities for medical management and mortalities
4 for surgical management on Table 20/395.

5 Q. Well, just so we can see
6 how we follow it up and then we'll go to some of the
7 other changes, this Table 27, for example, shows that
8 if the baby had had a ventricular septal defect,
9 there were 113 in that study, 20 of them would have
10 died if they were to be surgically managed within
11 the first 30 days.

12 THE COMMISSIONER: No, I think that
13 is percentage.

14 MR. SCOTT: I'm sorry.

15 THE WITNESS: 20 per cent.

16 MR. SCOTT: Q. 20 per cent would
17 have died and 23 per cent would have died in the first
18 year.

19 A. Yes.

20 Q. Is that an additional 23 or
21 does that include the original 20?

22 A. No. That's 23 per cent.

23 Q. And therefore if you take
24 all the defects that were surgically managed you
25 come out to a first year mortality rate of 40 per
cent averaged at the bottom?



DD3

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A. Yes.

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Q. Now, is there a table that

4

deals with infants who are not surgically managed?

5

A. I'm sure there is.

6

Q. Well, take your time.

7

A. I think Table 20 is probably
it. It is probably the easiest one to look at.

8

Q. All right. And there I take

9

it --

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MR. LAMEK: I'm sorry, what number?

11

MR. SCOTT: Table 20 at page 395.

12

Q. Again, the deformations are

13

listed under two headings, "Medical Management" and

14

"Surgical Management", and the averages for crude

15

mortality in each case is 40 per cent, which means

16

40 per cent die within the first year. Have I got
that right?

17

A. Yes. I can't remember what

18

the crude versus the adjusted, whether they excluded
certain things in the adjustment.

19

Q. All right.

20

Well now, apart from showing those

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gross mortality figures which you described earlier

22

in summarizing the reports as saying 35 to 40 per cent

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of babies with these deformations died in the first

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year, I then asked you if there were determinants that the report dealt with with respect to survival, and the first thing you dealt with was the severity of the anatomic diagnosis.

A. Yes.

Q. Now, does the report set up a system for measuring severity of anatomic diagnosis?

A. Well, the system they used is an arbitrary one and their conclusion that when they looked at the validity of their prognostic categories, as they call them, they were found to be reasonable but I don't know what the evidence is, the statistical analysis.

Q. Did the study tell you anything about the relation of mortality to the age of admission?

A. Oh, yes, that was a very important observation.

Q. And what did it tell you?

A. Well, that showed that the majority of deaths occurred in babies who were admitted during the first two months of life.

Q. And do you have the percentages?

A. I think it was somewhere around



DD5

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2 55 per cent or something like that. I can't see it
3 here.

4 Q. Well, can you take a moment
5 and put your finger on it in the report? We could
6 even take perhaps a short minute break if that is
7 necessary. You were good enough to give the figures
8 to me, I've got them but I wanted you to place them
9 in the report, if you can.

10 MR. LAMEK: Table 41, I think, isn't
11 it, Doctor?

12 THE WITNESS: I think that is probably
13 the one.

14 MR. LAMEK: Age of admission is
15 the determinant for survival.

16 MR. SCOTT: I want the percentages.

17 MR. LAMEK: It is given us the
18 percentages. 51 per cent of two months.

19 THE WITNESS: Table 41, I think
20 Mr. Lamek has given it.

21 MR. SCOTT: Q. All right. What
22 does that show?

23 A. It shows that in the first
24 two months the mortality is about 50 per cent; it is
25 49 per cent in one time span and 51 per cent in
another and then, after two months, the risk is lower,



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substantially lower.

Q. Does that mean that of the 40 per cent who will not survive the first year, 50 per cent of that 40 per cent will die in the first two months?

A. I haven't done the figures but I would think that it means that the greater contribution of the overall one-year figure is made in the first two months.

Q. Yes.

Now, is there a table that tells us the relationship between the age at which you submit to surgery and the mortality?

A. Yes, there is. Table 42.

Q. And what is the figure there?

A. Well, the figure there is that, in the first two months of life, the risk of dying from surgery is very much higher than after that age. The risk is somewhere around, again, 50 per cent for the first two months and about 20 per cent in the subsequent months of the first year.

Q. All right.

So, can I summarize it this way. In round figures, in dealing with cases of reasonably severe anatomical diagnosis, roughly 40 per cent of



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babies will not survive their first year of life?

A. Yes.

Q. Dealing with those same babies, roughly 50 per cent of them, whether they are surgically managed or not, will die in the first two months of life?

A. Yes.

Q. Now, is there a table that tells us the contribution of birth weight to the statistics?

A. Yes, there is. Table 43.

Q. Yes. And what does that tell us?

A. Well, that tells us that smaller babies have a higher mortality. They have used the cutoff point of 2 kilograms.

Q. You see, you have been saying for two weeks to Mr. Lamek that smaller babies are inclined to die faster and what I am trying to do here is to see, apart from your judgment, if there is evidence that sustains that and perhaps you can give me what the figures were in this study.

A. Well, the figures that they arrived at were that if you were less than 2 kilograms, the risk of dying before the first year was about



DD8

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50 per cent.

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Q. Yes.

4

A. If you were over 2 kilograms,

5

the risk of dying was 39 per cent, or 40 per cent.

6

Q. Yes.

7

A. Not a huge difference but it

8

is a difference.

9

Q. Is that a risk that is --

10

Is the weight factor a weight factor in this study

11

that is applied to a reasonably severe cardiac

12

malformation case?

13

A. I'm not sure. The 2 kilo-

14

grams is a bit lighter than I would have expected, but

15

the figures show that. I would have to get someone

16

to look at the material to see whether or not there is
that big a difference.

17

Q. Do you mean to say that you

18

would have thought that there would be more babies
dying who weighed less than 2 kilos?

19

A. No. I would have thought the

20

cutoff point might be a bit higher than 2 kilos.

21

Q. At what, say?

22

A. Well, say, at 2.5. But I

23

think that some biased statistician might be able to
look at that.

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Q. So that what we can say is that, on these three factors that we have looked at as applied to reasonably severe cardiac malfunctions, there is the production of a survival rate of 60 per cent for the first year, half of those more or less dying in the first two months?

A. Yes.

Q. Now, are there figures or comments in this report that deal with another factor; that is, extracardiac malformations?

A. Yes. They have a section on that which is on page 408.

Q. 8 or 80?

A. 408.

Q. Yes.

A. And the conclusion of that was that the presence of an additional major malformation is usually a direct affect of survival.

Q. Does the report - I interrupted you - tell you anything about the extent to which an extracardial malformation is likely to be found in a baby with a cardiac malformation?

A. Yes, I believe there is a figure on that, and I don't know where that is. This group has published separately many, many papers on



DD10

1
2 the association of congenital heart disease with
3 extracardiac malformations, and the usual figure that
4 is given for this is somewhere between 25 and 30 per
5 cent.

6 Q. All right. Now, what does
7 that mean exactly? If you take a baby who has a
8 cardiac malformation, does that tell you anything
9 about the likelihood of another system being mal-
10 formed?

11 A. Yes, because the figure is
12 somewhere around 25 or 30 per cent. That means that every
13 clinician who is concerned with a baby who has
14 heart disease is looking to see whether there is an
15 additional anomaly.

16 Q. And he will know that, based
17 on those figures that, say, in one of three cases
18 there will be an extra anomaly?

19 A. Yes.

20 Q. And are there any figures
21 in the report that deal with mortality when there is
22 more than one anomaly in a system? You have the
23 cardiac anomaly and then you have the 30 per cent,
24 I think it is actually 28 per cent where there is
25 an extra anomaly. What does that do to the death
rate?



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A. Well, I'm not sure about the rise in numbers. You may have missed that, but there is no question that with very severe anomalies, very severe additional extracardiac anomalies, the mortality is gravely affected. In fact, in this study, they excluded those patients from the mortality conclusions by calling them an adjusted mortality after they had excluded those patients. But I can't find the spot specifically that, whether if you have five, you have more risk than if you have two.

Q. Yes. Well, is there any sense in the medical profession on that subject?



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A. I think the more important thing is the nature of the extra cardiac anomaly. If you have only one major additional anomaly and it happens to be in the brain then that's much more important than if you have a turn in the little left finger.

Q. Let's take each of those subjects, and I want to ask you first of all about the severity of cardiac malfunction. This report makes an effort as you have described to gauge the severity of the malfunction.

Now, you have given evidence, Doctor, that in the 36 babies that were examined by you and Mr. Lamek, I think all but three babies Pacsai, Hines and Hayworth exhibited a congenital structural abnormality of the heart?

A. Yes.

Q. So of the 36 I think you were looking at 33 had a cardiac malfunction of some type?

A. Yes.

Q. Now would you be able to gauge within reasonable medical limits the severity of those 33 heart defects?

A. Yes.



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Q. Can you tell the Commissioner how you would approach that exercise of measuring the severity, because when we know their severity then we can take the New England figures and see how many of them would have died had they lived in New England. What is the process by which you would gauge the severity of those malfunctions, cardiac malfunctions?

A. Well you would look at the - I think the cornerstone of that would be the anatomic abnormality.

Q. Yes.

A. The precise definition of the abnormality in the heart itself.

Q. Yes.

A. Because there are certain malformations in whom the delineation of the detail tells you immediately that this is such a severe malformation that death is inevitable.

Q. Yes.

A. So that would be an example of one extreme in the situation.

In another you might find a baby who has a ventricular septal defect which might be of moderately large size. So you would be able to say



1
2 that the anatomy was moderately severely distorted
3 and you could tell, depending upon the age of the
4 patient how well that heart was functioning that you
5 could make predictions as to the size of the defect
6 in relation to the size of the child, what the probable
7 outcome for that baby might be. Not in terms of
8 immediately whether it is going to die or anything
9 but in terms of the prognosis generally.

10 Q. Well, I'm going to ask you
11 to do this in due course, but I just want to make
12 sure that you think you can do it.

13 If you take the New England statistics
14 that in moderately severe cases 40% of babies with
15 moderately severe cardiac malformation do not survive
16 one year?

17 A. Yes.

18 Q. If you take that figure, do you
19 think you can characterize the babies about which we
20 have been talking with sufficient certainty to apply
21 that figure from the New England standard to them?

22 A. I think you probably could.

23 THE COMMISSIONER: Would this be a
24 good time?

25 MR. SCOTT: Yes, Mr. Commissioner.

THE COMMISSIONER: We will take



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15 minutes then.

---Short recess.

---Upon resuming.

THE COMMISSIONER: Yes, Mr. Scott.

MR. SCOTT: Thank you, sir.

Q. Let me come back, Dr. Rowe, it's just occurred to me to ask you to do something that probably is not possible to do. You told us that the New England report showed that of the babies in their study 35% to 40% did not survive the first year, have I got that right?

A. That is correct.

Q. You also told us that age and admission had a bearing because 54% - I'm sorry, 51% of those babies died in the first two months of their life.

THE COMMISSIONER: That is 51% of the 40%?

MR. SCOTT: Yes.

THE COMMISSIONER: That is from which table?

MR. SCOTT: I haven't got the table numbers.

MR. SOPINKA: Table 41, page 407.

MR. SCOTT: Q. You also told us



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that the study revealed that extra cardiac malforma-
tions occurred in about 30% of the babies, is that
right?

A. I couldn't find their figure
you recall.

Q. All right. Well, we can leave
that, that was your assessment.

A. My understanding was it was
somewhere around there.

Q. And I think you also told us
that one of the tables show that 56% of the babies
died, if operated on, in the first two months?

A. Yes.

Q. And the last thing you told us
was that the ---

THE COMMISSIONER: I'm sorry, that
is Table 42 I guess, 56%?

THE WITNESS: I think so.

MR. SCOTT: Q. Yes, it is, sir.

A. That is 50% if you take both
time periods.

Q. And then you also told us
that the report presented I think at Table 39, a
method for measuring the severity of cardiac malforma-
tion?



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A. It is an arbitrary method,
yes, I think it has some problems but it is reasonable.

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Q. It fundamentally divides the
cardiac malformations into various categories and
groups them with respect to severity moving from
Group 4, which is the most severe to Group 0 which
is the least severe, is that right?

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A. Yes.

THE COMMISSIONER: What is that table
please?

THE WITNESS: That is Table 39.

MR. SCOTT: It is a linear table
rather than a graph.

THE COMMISSIONER: Yes, I see.

MR. SCOTT: Q. And then you also
gave us figures and I don't again have the table
number, on birth weights.

MR. SOPINKA: Table 43.

MR. SCOTT: Q. Table 43 I am told
that that affects mortality.

A. Yes.

Q. Now then, I asked you if you
thought you could take the 36 cases which you have
discussed with Mr. Lamek and measure the severity of
cardiac malformation applying generally the standard



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that was used in the New England report, and I think you indicated that you could do that?

A. Yes.

Q. And is it possible, in your judgment, having done that to make a reasonable judgment as to the severity of cardiac malfunctions in those cases?

A. Yes, it would be possible to do that, in two ways. One to do it on the condition prior to death.

Q. Yes.

A. And the information available to us prior to the death of the patient, and then it could be done with slightly different results from those of whom we have information available in post mortem.

THE COMMISSIONER: I am sorry, I am having trouble, I am just not sure what it is he is going to do.

MR. SCOTT: He is not going to do anything yet, I am just going to ask him if he can measure the severity of those, of the 36 babies ---

THE COMMISSIONER: Yes.

MR. SCOTT: --- applying the New England, roughly the New England standard for severity.



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THE COMMISSIONER: That is Table 39?

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MR. SCOTT: Q. Yes. If he can make

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that judgment with a reasonable degree of confidence

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about 36 babies who died, and I want him to make that

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judgment before their death. That is to say I don't

7

want him to look at the autopsy. I take it Dr. Rowe,

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that you may, it is conceivable that you might make

9

a judgment that a baby's formation was not particularly

10

severe only to find after autopsy that you were wrong

11

and that an examination of the heart showed it be

12

A. It would be most likely in

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that area that there would be a difference.

14

Q. So what I wanted to know,

15

whether you could do it with any confidence, is judge

16

how you would rank the babies with regard to severity

17

of cardiac malformation before their death. We have

18

spent a lot of time looking back and second guessing

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what we might have thought. I want you now to begin

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to make judgments as you would have made them in the

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THE COMMISSIONER: Are you doing this

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for its own sake or is this leading to something?

23

MR. SCOTT: It is leading to something.

24

THE COMMISSIONER: Have you told us

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what that something is yet?

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MR. SCOTT: It is going to show that the judgment of the cardiologists as to severity of these illnesses led to a conclusion that a number of the babies might die.

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THE COMMISSIONER: Oh, I see. The idea being if all 36 of them came in diagnostic Group 4 it would be a reasonable assumption that they might all die, is that it?

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MR. SCOTT: Well, to be frank, one of the points I think Mr. Lamek has made, and particularly with respect to the September conference is you had 10 babies who died in July and August.

THE COMMISSIONER: Yes.

MR. SCOTT: That graphically is more than you had in April and May, why didn't you do something about it? In fact I think that is the word Mr. Lamek used. The answer to that question is governed by how you looked at those deaths, if you regard them as severe cardiac cases in which death was 70% risk then you have an elevated level of deaths, but you have another high point like one of the dozens of graph but it is a great misfortune.

THE COMMISSIONER: But surely he can use the autopsy results if they were available in



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2 September, could he not?

3 MR. SCOTT: Well, I will ask him
4 to do that in due course, but I have to take it stage
5 by stage.

6 THE COMMISSIONER: All right.

7 MR. SCOTT: I am very slow about it.

8 THE COMMISSIONER: If you wanted
9 to have a prediction if it is for the purpose of
10 showing the propriety of his actions in September
11 and December or January, because then he surely should
12 have available to him all the information he had
available at that time.

13 MR. SCOTT: Yes, we will come to
14 that.

15 THE COMMISSIONER: Yes, all right,
16 I am sorry.

17 MR. SCOTT: Thank you, sir.

18 Q. Now the second thing is extra
19 cardiac - first of all we have established that you
can make that judgment about severity, am I right?

20 A. Yes.

21 Q. Now let's come to extra
22 cardiac malformations and I will find it for you
23 later in the New England report. My understanding is
24 that 28% of their babies exhibited additional
25



EE11

malformations beyond heart.

I want to ask you, first of all,
if you can ---

MR. LAMEK: I just wanted to tell
you that was page 392.

MR. SCOTT: Q. It is really Table
18, isn't it, Dr. Rowe?

A. Yes.

Q. It shows the relation between
extracardiac anomalies and diagnostic categories
running from 63% down and averaging at 28%. I take
it for the babies in the - among the 36, you can
make a judgment about the extent to which they
exhibited extracardiac malformations.

A. We can.

THE COMMISSIONER: This figure,
this Table 18 if I understand it correctly does that
say an average around 28% of babies with cardiac heart
defects of one sort or another have extracardiac
anomalies?



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MR. SCOTT: That is the way I
read it. Perhaps I had better ask Dr. Rowe.

THE WITNESS: That is correct.

Q. And I think you have also
told us - it's at page 394 --

THE COMMISSIONER: But before we
go into this, I don't understand it. Table 18,
endocardial cushion defect. I am not too sure - in
fact, I am sure I don't know what that stands for,
but they say 63% of the total. What is that total of?

THE WITNESS: 63% of 119.

THE COMMISSIONER: Oh, I see. The
119, that particular defect?

THE WITNESS: Yes.

THE COMMISSIONER: 63% of them had
extracardial defects.

THE WITNESS: Cardiac anomalies.

THE COMMISSIONER: I see.

THE WITNESS: The reason for that
very high figure there, Mr. Commissioner, is that the
majority of patients with endocardial cushion defect
have Down's Syndrome so this high association.

MR. SCOTT: Q. The significance
of this, Dr. Rowe, is revealed at page 394 where the



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2 report says, if I read it correctly, second sentence,
3 "effect on survival":

4 "It was shown that for infants with
5 severe extracardiac anomalies there
6 was a significantly higher mortality
7 than for infants without associated
8 anomalies."

9 A. Yes.

10 Q. So that if you had only a
11 cardiac malfunction, your chances of surviving the
12 first year or of dying the first year was 40%. If you
13 had a severe extracardiac anomaly on top of that, your
14 chances of dying went up to 48%.

15 A. Yes.

16 THE COMMISSIONER: These are not
17 figures you are getting from the table? These are
18 figures that you are --

19 MR. SCOTT: I am getting from the
20 text of the study at page 394.

21 THE COMMISSIONER: Yes, I see.

22 MR. SCOTT: Q. And can I ask you,
23 doctor, is that consistent or inconsistent with your
24 general observation in the clinic or in the hospital
25 about the effect of extracardiac malfunction?

A. I think what they are doing



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there, though, is they are accepting as -- they are accepting only the highest grade of extracardiac malformations as having a major influence, and they say without associated anomalies.

Unless I am mistaken that means the milder or more moderate forms of malformation.

I think those things may have an additional effect or importance on occasion as well, but that is the data from their study and the way they have set it up, so I am prepared to accept that.

For example, if I may amplify on that just to get the point?

Q. Yes.

A. The baby who has tetralogy of Fallot and who has a bad bilateral harelip and cleft palate would not be graded in this study as being of high risk.

Q. What would you say?

A. I would think this is a high risk situation because the baby who has got tetralogy of Fallot and is therefore blue is going to have a great deal of difficulty with a lot of mucus in the throat and constant suctioning so this is the sort of baby that is very liable to have an airways obstructive type of problem that could very well be critical to



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2 survival.

3 So I wouldn't entirely agree with
4 this. I agree with the broad sweep of it. They are
5 talking about a major associated anomaly like a
6 trisomy 18, a chromosomal defect in which the survival
7 of the baby in any event is just a matter of a month
8 or two.

9 Q. So the report is more
10 conservative than you --

11 A. I would have thought so.

12 Q. They take into account in
13 moving the figure from 39% to 48% only as you have
14 said severe extracardial malfunction?

15 A. That is the way it appears
16 to me, yes.

17 Q. What would you say about
18 the less than severe extracardial malfunction as playing
19 a role in mortality of babies with cardiac malfunction?

20 A. I think obviously mild extra-
21 cardial anomalies, like an extra toe or something like
22 that, are not going to have any effect but I think
23 there are a number of more moderate group of mal-
24 formation, of extracardial malformations, that might
25 conceivably have quite an effect.

Q. Can you just tell the



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Commissioner what they are in your opinion.

A. Principally conditions which affect the respiratory system. The cleft palate and harelip is one. Trachialesophageal fistula; diaphragmatic hernia is another. In other words there are a number.

Q. Can you list any others --

A. I can formulate a list for you if I sat down and thought about it.

THE COMMISSIONER: These are a list --
Are these the severe --

MR. SCOTT: No, these are --

THE COMMISSIONER: Table 19 seems to list -- I take it Table 19 are the ones, are they not?

THE WITNESS: Yes.

THE COMMISSIONER: Those are the ones they do take into consideration, do they not, Table 19?

THE WITNESS: They just give a broad sweep, but if we kept to that sort of list, Mr. Commissioner, it would mean respiratory anomalies might not be graded by the New England study as severe. But they I think -- in my view, respiratory anomalies can be important in prognosis.



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MR. SCOTT: Q. The point I want to get at is when the New England study moves the death rate from 39% to 48% because of a severe extracardial malfunction, you have said that your reading of the report leaves them to define severe extracardial malfunction by excluding malfunctions that you would have included as having an effect?

A. As maybe having an effect, yes. There is a risk factor which you would have to assess. I think they are doing a broad sweep and I wouldn't quarrel with the way they have done it but I think there are definite situations where the presence of a less than severe extracardial malformation can have a significant effect on the outcome.

Q. Let me turn to one other factor.

THE COMMISSIONER: Before you go any farther, I am not - and please don't misunderstand. I am not arguing with you. I am trying to get it through my head where you are leading us. It would seem to me that it would naturally follow that a baby who was sick with heart disease of some sort, if he had something else wrong with him, it certainly wouldn't be a help. It would be worse.

Now, I can well understand that the



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more serious it is, perhaps the more likely it will make that baby die. But what is all of this leading to? Are you going to try to transpose these percentages in some way on to the 36 babies that we are investigating?

MR. SCOTT: What I seek to show, Mr. Commissioner -- let me put it this way: What has been said is that in the epidemic period there was by graph an elevated level of deaths.

THE COMMISSIONER: Are you going to try to show to us in the epidemic period there was also extracardial defects?

MR. SCOTT: What I am going to try to show to you is that the babies who died in the epidemic period - this has nothing to do with whether there was an intentional homicide; I have nothing to say about that at the moment - but the babies who died in the epidemic period were very high risk babies.

THE COMMISSIONER: But there may have been the same kind of babies in the other --

MR. SCOTT: There may have been. If my friend shows that or somebody else shows that. I am simply showing --

THE COMMISSIONER: It doesn't tell me anything unless I know what the other babies were



FF8

as well.

MR. SCOTT: Well, it may be that someone will tell us about the other babies.

THE COMMISSIONER: You see --

MR. SCOTT: It may be that even I will tell you about that but what I seek to show you now is -- well, let me put it this way: Mr. Lamek's questioning - I almost said evidence - was advanced on the theory that you had a baby who was stable, who was about to, in some cases, the way he put it, you thought was about to be sent home and all of a sudden - his language; not mine - all of a sudden there was an incident and the baby died.

THE COMMISSIONER: Yes.

MR. SCOTT: Now that bears examination. I have gone at it one way by showing the indicia of suddenness is not to be relied upon. I think it is also helpful to show you, sir, if I can, that these babies who died were to a very high degree likely to die because of the conditions, and when Dr. Rowe said that to Mr. Lamek, Mr. Lamek challenged him; wouldn't accept that, and that of course is what this evidence is led to.

THE COMMISSIONER: Well, all right. Thank you. I am having some difficulty in putting



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together the New England figures with our own figures.
Maybe I am just misunderstanding.

MR. SCOTT: We don't have those
figures, sir. For example, if you take the New
England figures that show 50% of the babies defined
in a certain category died within the first year --

THE COMMISSIONER: Yes.

MR. SCOTT: -- we can only produce
the comparable figure by analyzing all the babies,
hundreds, who went through the ward in the relevant
period of time. And then we would have a figure that
was the same, more or less, than the New England
figures.

We might indeed have a figure that
showed that our mortality rate at the Toronto Sick
Children's Hospital was substantially less than 48%.

We haven't done that exercise, and
it is an exercise of very considerable scope, and I
am not sure that we are going to be able to do it. So
short of that, what I am saying to you is I think it
can be demonstrated that these babies, many of them,
not all of them, were very severely ill and likely to
die.

If everybody accepts that proposition,
I have nothing more to say.



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THE COMMISSIONER: Oh, no, but I don't think I can make that offer to you that everybody does accept the proposition. Certainly, though, Dr. Rowe has given us evidence that these babies were suffering from certain cardiac and other malformations as a result of which their deaths were not surprising.

What I am trying to find out is how we are particularly helped by the New England report.

MR. SCOTT: You are helped by the New England report because the first thing it shows is that babies in this category -- Dr. Rowe has been saying --

THE COMMISSIONER: Babies with extracardiac problems would die more likely more quickly than babies with just cardiac problems.

MR. SCOTT: What it shows is that almost 50% of babies with these difficulties in children's hospitals in New England, which includes the Boston Children's Hospitals, one of the great hospitals of the world, 50% of them die. Now, that it seems to me is a fact that is demonstrated and the variables that can be introduced, such as extracardial malfunctions and so on, show how those extra anomalies add to the figure.

Now the first thing we begin with



FF11

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2 is that there is no suggestion that anywhere near
3 50% of the babies in Toronto Sick Children's Hospital
4 died in this relevant period. Our percentage is
5 way below that. No doubt about that.

6 We have a ward that will hold 38
7 over the epidemic period. So our gross figures are
8 different than the New England figure.

9 Then you take the group of babies
10 who regrettably died and you look to see whether they
11 were, because of their malfunctions, candidates by
12 virtue of their conditions and the indicia that the
13 New England study reveals, candidates to die within
14 the first year.

15 Now, I hope that is helpful and if
16 it is not I don't want to...

17 THE COMMISSIONER: No. I'm sure
18 it is helpful, Mr. Scott, but I think you must under-
19 stand that all I am doing is trying to make sure that
20 your brilliant advocacy is getting through, that is all,
21 to me, and so to understand --

22 MR. SCOTT: Well, as long as your
23 lordship and I understand each other we don't have to
24 worry about the others. Let them look after them-
25 selves.



/BB/ak

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2 THE COMMISSIONER: Well, my ignorant
3 questions might help them to avoid some of their own.
4 So, we'll see.

5 MR. SCOTT:Q. Well, let me ask you,
6 Dr. Rowe, if you can refer now to page 396 of the
7 report. Under High Risk Factors is there a reference
8 there to the size and age of babies at time of birth?

9 A. Yes, there is.

10 Q. And what does it say about
11 that risk?

12 A. It says that low birth weight
13 was found to affect survival regardless of all
14 other factors.

15 Q. All right. Now, that's the
16 third factor.. We've had the severity of cardiac
17 *malformation* renal function, we've had the presence of extra
18 anomalies, we've had the size and age of babies at
19 the time of birth. Now, I want to ask you about the
20 failure to grow and thrive in weight and height and
21 I want to ask you if that terminology refers to a
22 phenomenon that is recognized in cardiology?

23 A. Yes.

24 Q. And what does it mean?

25 A. This means that babies don't
grow properly. They don't grow, they don't put on



weight, and they don't elongate.

Q. And what is the impact of that in terms of a baby with these other deficiencies, cardiac anomalies and additional anomalies?

A. Well, the most obvious feature is that they're very thin and skinny and they have very little reserves in terms of fat or any of the usual energy reserves that babies have. So, they are at high risk for running short of fuel, as it were, if they are stressed in any further way; stressed by infection or stressed by an arrhythmia, stressed by progression of the disease and so on.

Q. All right. Now, with respect to these four factors, did you at my request make a review of all 36 babies?

A. Yes.

Q. Yes. And did you review those babies with the four factors I have elicited in mine?

A. Yes.

THE COMMISSIONER: Those four factors, so that we all know what they would be.

MR. SCOTT: The severity of cardiac malformation.

THE COMMISSIONER: And the non-cardiac.

MR. SCOTT: The presence of the



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non-cardiac anomaly.

THE COMMISSIONER: Low birth weight.

MR. SCOTT: Size and age and time of
birth and failure to thrive.

THE COMMISSIONER: It would be size
and weight, not age.

MR. SCOTT: Thanks.

THE COMMISSIONER: And the failure
to grow and survive, yes.

MR. SCOTT: Yes.

THE COMMISSIONER: So, that's cardiac,
non-cardiac, birth weight and growth weight.

MR. SCOTT: Q. Now, have you got that
in printed form?

A. Yes, I have.

MR. SCOTT: Now, I don't have copies
of this for everybody at this stage, so, we can
undoubtedly get it.

THE COMMISSIONER: Well, it's pretty
close to closing time anyway, so, we could - I don't
know, it will probably take some considerable time to
go through this, would it not, for each one of these
children? It might be an idea to have copies made.

MR. SCOTT: Perhaps I can ask some
questions about it that won't need the document.



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THE COMMISSIONER: No, no, by all
means, yes.

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MR. PERCIVAL: Unfortunately,
Mr. Commissioner, the questions are going to mean
nothing to the rest of us.

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THE COMMISSIONER: Well, that's
what I thought.

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MR. SCOTT: But I said I thought I
could ask some questions without the document.

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THE COMMISSIONER: Mr. Scott has
promised to ask good general questions that are going
to mean a great deal to us.

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MR. PERCIVAL: We wouldn't need the
document you mean?

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THE COMMISSIONER: We won't need the
document at all. I promise, I won't even look at
mine so that I will know when ---

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MR. LAMEK: Now you're in trouble,
Scott.

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2 THE COMMISSIONER: Well, if we copy it
3 we may as well rise for the day but if you can ask
4 some general questions that will be of assistance, let
5 us have them.

6 MR. SCOTT: Q. Well, first of all,
7 Dr. Rowe, do I understand that this study was prepared
8 in the last couple of weeks?

9 A. Yes, it was.

10 Q. And it was prepared in the
11 light of the analysis in the New England Journal, is
12 that correct?

13 A. Yes, it was, with the exception
14 that the severity of malformation was handled a little
15 differently on a number of additional factors.

16 Q. But it is with that proviso
17 an attempt to model an analysis on the New England
18 study?

19 A. Yes.

20 Q. And do I understand that at my
21 direction, perhaps unwisely, I have to take the
22 responsibility for this, I think it was prepared as if
23 you were looking at the patients before their death.

24 A. Yes.

25 Q. And do I understand that Dr.
Freedom was also asked to do the same thing?

A. In respect to the predictions,



GG2.2

BB/wb

yes.

Q. Yes. And that you and he did not compare your analyses before they were completed?

A. No.

Q. Now, perhaps I will have to start looking at the form now, Mr. Commissioner, if you want to stop.

THE COMMISSIONER: I take it we're not going to have Dr. Freedom's document until we have Dr. Freedom. I take it he has a different document, he's prepared a different document.

MR. SCOTT: Well, he did the same exercise, applying the New England Study to this exercise. He did it and we asked him to do it alone, we didn't want Dr. Rowe and Dr. Freedom comparing notes as they went along. My friend can have his result anytime he wants it.

THE COMMISSIONER: That's fine. No, no, no reason to use it now. I think we could probably, if there is nothing else you want to ask until we go over to the document itself, well then, let's --

MR. LAMEK: Mr. Commissioner, can I suggest we copy it here so Counsel can take it away with them. We can take it up to the 22nd Floor and



GG2.3

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2 have it copied immediately.

3 THE COMMISSIONER: All right. Well,
4 I think Counsel can just drop in on their way home.
5 It's a funny way to go home, but they can do it, go
6 up to the 22nd Floor.

7 MR. LAMEK: I go home that way every
8 night.

9 THE COMMISSIONER: All right. Well
10 then, until 10:00 o'clock tomorrow. Have you any
11 thoughts for us, Mr. Scott, of how long you think
12 you might take?

13 MR. SCOTT: A full day.

14 THE COMMISSIONER: But just all day?

15 MR. SCOTT: Mr. Lamek was complaining,
16 he was telling some of our colleagues and associates
17 in the press that we were getting very far behind and
18 he was alarmed to hear that the 12 other Counsel
19 might take two weeks, which was, of course, just a
20 third of what he had taken. So, he encourages us to
21 be as brief as we can to leave more time for him.
22 But we'll move along as quickly as I can.

23 THE COMMISSIONER: There's a problem
24 Mr. Sopinka mentioned to me that apparently he is not
25 available on Thursday. So, after you is Mr. Ortved
and after Mr. Ortved, I guess, is Mr. Strathy.



GG2.4

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3 Mr. Strathy, you are prepared to
4 proceed?

5 MR. STRATHY: Yes.

6 THE COMMISSIONER: If time is
7 available. I don't know, Mr. Hunt, are you mixed up
8 with Mr. Sopinka's motion?

9 MR. HUNT: No.

10 MR. SOPINKA: They've got so many
11 people over there.

12 THE COMMISSIONER: Well, I know, you
13 work on quality.

14 And then, after Mr. Strathy, you will
15 be prepared.

16 MR. HUNT: I would doubt whether that
17 would be on Thursday in any event, just by the time.

18 THE COMMISSIONER: Well, no, but if it
19 is I just would like you to be prepared. That may be
20 all we'll get through on Thursday but it may not.

21 However, you haven't any thoughts on
22 how long you'll be?

23 MR. STRATHY: If I'm reached on
24 Thursday I would be very surprised if I was finished
25 on Thursday.

THE COMMISSIONER: Yes. But don't
give Mr. Hunt too much comfort or he won't be



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prepared.

MR. STRATHY: No, I wouldn't do that.

THE COMMISSIONER: All right.

MR. SOPINKA: Thank you, Mr.

Commissioner.

--- Whereupon the hearing adjourned until Wednesday,
August 17th, 1983 at 10:00 a.m.

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